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Section of Comparative Medicine.

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DISCUSSION ON THE COMPARATIVE PATHOLOGY OF TUMOURS.

Dr. William Cramer: Cancer is distributed throughout the vertebrate kingdom and exhibits the same fundamental features in animals as it does in man. It arises *de novo* in each individual, begins as a local disease and spreads by metastatic dissemination. It is not contagious. The disease cannot be transmitted from an animal of one species to an animal of another species or even of the same species, for the transplantation of tumours, which is restricted to animals of the same species, is not a transmission of the disease but merely an *in vivo* culture of malignant cells, inoculated animals serving as the medium nourishing the transplanted cells. The only exceptions to this rule are some of the tumours in the fowl, where the disease can be initiated *de novo* in a normal animal by the inoculation of cell-free extracts.

One feature common to cancer in animals and in man, which is essential for an understanding of the distribution of the disease in the animal kingdom, is the characteristic age-incidence of cancer. Cancer in man is a relatively rare disease—1 case in 1,000 living persons. In children and young adults it is extremely rare and it becomes increasingly infrequent as age advances. In order to establish the presence or absence of the disease in any particular species, it is therefore necessary to examine a sufficiently large number of middle-aged and old individuals. When this condition has been observed, no species has so far been found which was free from cancer.

The reason why cancer has been noticed less frequently in most species of animals than in man is due to two main causes. In the case of wild animals living in their natural habitat there is the obvious difficulty of making observations on a sufficiently large number of middle-aged and old animals. When that has been possible—for instance in fishes where many thousands of fishes have been examined—malignant new growths have been found in considerable numbers. The apparent rarity of cancer in many species of domestic animals finds an explanation in the fact that these animals are killed, as a rule, before they have reached the cancer age. In those species of domestic animals which are allowed to live until they are middle-aged or old, such as the fowl, the cat, the horse, and especially the dog, cancer has been found with a frequency roughly proportional to the number of middle-aged or old animals which come under observation.

While cancer has the same fundamental features in all vertebrates, there are variations in the different species in certain details. In some species the sexes are affected with a different frequency; in some species epithelial tumours are more

frequent than sarcomata, in others the relation is reversed; again, the organ incidence varies greatly in different species. The explanation of these differences is found partly in a constitutional difference in the tendency to malignant development residing in the different tissues and organs of different species. It may also be due to the differences in local conditions giving rise to various forms of chronic irritation, which lead to cancer. The established association between certain gross parasites and cancer, which varies from species to species, also plays a part. [ABSTRACT.]

Dr. S. L. Baker: *Tumours of fowls.*—The tumours of fowls occur sporadically in the same way as do those of mammals.

There is no evidence of infectivity. Among many hundreds of fowls bearing Rous Sarcoma No. 1 housed with normal fowls in very close quarters, I have only once seen the transference of a tumour to an uninoculated bird. This was almost certainly a direct inoculation by a peck as there was in the pen a fowl with an ulcerated tumour which had been pecked by the other fowls.

The types of tumours occurring among fowls are, as far as I have observed, the same as those of mammals. I have seen the following types: Fibroma; spindle cell sarcoma (several types); hypernephroma; columnar-cell carcinoma of intestine; squamous-cell carcinoma of epidermal origin; teratoma of ovary; ganglioneuroma; giant-cell tumour of bone.

A renal embryoma, resembling Wilms' embryonal tumour in the human, has been reported.

The sarcomata.—Many types have been described, those that have been successfully transplanted have been found to be filterable. It appears very probable that all fowl sarcomata are filterable.

The carcinomata.—No one has ever transplanted a fowl carcinoma successfully for any number of generations and, therefore, nothing is known as to the filterability of these tumours.

Filterability.—In the case of sarcomata the conveyance of the tumour-producing agent by tumour extract, which had been passed through a Berkefeld filter, was originally demonstrated by Peyton Rous and has been amply confirmed by numerous subsequent workers. The infective agent is highly specific both as to host and as to the cell type of the resulting tumour.

This agent exhibits the properties of a virus in that: (*a*) It is destroyed by antiseptics and by moderate heat; (*b*) it can be preserved in dried material and in glycerinated material; (*c*) it is potent in fairly high dilution (1/1000 of Berkefeld filtrate will produce tumours in many cases).

Its action appears to be entirely local, but differs from that of chemical carcinogenic agents in that: (*a*) It is highly specific as to species and often even as to breed of host; (*b*) it acts only on one type of cell—the fibrocyte in the case of Rous Sarcoma No. 1; (*c*) there is no preliminary pre-cancerous alteration of the tissues; (*d*) the action is very rapid—a large tumour being produced in about two weeks in many cases; (*e*) the agent increases in virulence by passage through a number of hosts.

For these reasons it appears to me very probable that we are dealing with a living virus and not with a non-living chemical product.

Assuming that the fowl sarcomata are caused by a filterable virus, it still remains uncertain whether fowl carcinomata have a similar ætiology.

Although the relationship between the fowl carcinomata and mammalian cancer is, so far, obscure, these tumours at least demonstrate the fact that a filterable agent is capable of causing a neoplasm; to this extent they support the possibility of an extrinsic virus as a cause of cancer.

Dr. Leslie Sheather: I do not claim to have made a special study of the tumours occurring in the lower animals, but naturally a certain amount of information has been gathered in the course of diagnostic work done in veterinary pathological laboratories. It seldom happens that a full account of the case is sent when a tumour is submitted for examination, consequently a considerable amount of material is more or less "lost."

It must not be forgotten that there is one fundamental difference between human and veterinary medicine, namely, the intrusion of the question of the cost into practically every branch of the latter. Detailed investigation is expensive, and if it is to be carried out, some provision must be made for financing it. While financial support presents some difficulty, there is a difficulty which is possibly greater. In the human subject life may be terminated by disease, accident, or old age. In the lower animals, and particularly those used for food, slaughter takes place, in the immense majority of cases, before the age for cancer formation is reached. In the case of the companions or pets of man—the horse, dog, and cat—there is a greater chance of long life.

Middle life appears to mark the beginning of the period at which cancer may occur, but in the lower animals—as in man—cases occurring earlier than this are not unknown. I have encountered many cases of cancer in dogs under four years of age, and in my own laboratory recently I have had a case of what appears to be multiple sarcoma-formation in a three-year-old cow.

Searching my memory (as the notes of the cases I encountered before 1927 are not available) and turning up my records of the last four years, I am able to give the following details regarding the types and distribution of tumours occurring in the lower animals.

Sarcomata.—In the horse these are encountered, usually as a multiple development, in the subcutaneous tissue. Multiple sarcoma-formation sometimes occurs in the small intestine also.

I have encountered multiple sarcoma formation in the cow, and also cases with secondaries in the lungs.

Sarcomata are common in the dog, and I have records of their occurrence in this species involving the skin, lip, mammary gland, tongue, gums, liver, intestines, spleen, membrana nictitans, and kidneys. In the dog secondaries in the lungs are not uncommon. I have not seen so many cases of sarcoma in the cat as in the dog.

Carcinomata.—A not infrequent site of carcinoma in the horse is the eye, with secondaries in the glands. I have encountered cases involving the tongue and the penis.

Carcinoma does not appear to be very common in the cow, but that is possibly due to the factor already mentioned, namely, the occurrence of death at an earlier age. I do not know that any case of carcinoma of the mammary gland of the cow has been recorded. I have seen several cases in which the skin has been involved.

In the dog this type of tumour involves the skin, gums, liver, mammary gland, and testicle.

In the sheep in Australia a form of cancer occurs which involves the ears. This was described by Dodd in the *Journal of Comparative Pathology* in 1923.

Dr. J. R. M. Innes: It is not possible to make a strictly scientific comparison between neoplastic diseases in domesticated animals and analogous conditions in man, since statistical data regarding the incidence of the different types of tumours in the various species are not sufficiently extensive, and there are many types of tumours in man which have not yet been shown to occur in animals. While tumours

of the lower animals are perhaps of relatively minor importance, it cannot be doubted that the same laws govern their origin and formation as in man. Comparative oncology is thus a matter of practical importance and not merely one of academic interest.

The following table, which has been compiled from a series of cases examined within the last few years, affords an indication of the types of tumour which occur in the domesticated animals, but the number collected is not large enough to indicate the influence and relation of age, sex and other factors. The growths have been obtained from the dog, horse, ox, sheep and cat.

Fibromata	17	*Epithelial tumours :—*	
Chondromata	1	Adenomata	4
Osteomata	4	Adeno-chrondromata	2
Osteo-chondromata	4	Adeno-carcinomata	9
Myxomata	1	Fibrocystic adenomata (testis of dog)	2
Lipomata	3	Papillomata	9
Angiomata	14		
Leiomyomata	2	*Carcinomata :—*	
		Squamous-cell,	22
Sarcomata :—		Basal-cell,	—
Round-cell,	7	Transitional-cell carcinomata or lympho-epithelioma	1
Fibro-sarcomata,	8	Embryonal carcinoma or seminoma. (Testis of dog)	5
Osteogenic	—	Liver-cell carcinoma or hepatoma	1
Gliomata	—	Dural endothelioma, psammoma, or meningoma	1
Melanomata	18		
Lympho-sarcomata	2		

Total = 137

Fibromata.—Most of these were located in the skin and subcutaneous tissues, other sites being the vagina, tail, prepuce and ear. Considerable structural variation was encountered, from the soft cellular type with little stroma, to extremely dense hard growths in which collagen was abundant. *Osteomata.*—Included in this group are fibrous subcutaneous growths in which bone was present, but it is questionable whether these should be regarded as neoplasms or as examples of metaplasia. It was difficult to determine whether in these tumours the bone was the essential part of the growth or merely the secondary product. It has been observed that such growths in the horse arise as a result of trauma and chronic inflammation. *Osteo-chondromata* are of frequent occurrence in the mammary gland of the bitch, sometimes with a concurrent adenomatous process; their benign nature is well recognized. *Lipomata.*—These are frequently observed in slaughtered cattle in abattoirs, the common sites being the intestine and mesentery. *Angiomata.*—These include the cavernous and plexiform type, most of the former being located in the liver of the ox in which they are very common. The plexiform type were mainly located in the subcutaneous tissues of the dog. *Leiomyomata.*—These growths were extirpated from the uterus of a bitch, one case being in the nature of small multiple nodules. No case of rhabdomyoma has been encountered. *Sarcomata.*—These were located in the skin and subcutaneous tissues and, in one case, in the nasal cavity. In two instances metastases were present in the liver, the primary growth being in the skin. The same variety of cell types has been observed as in man, the small round cell, large round cell and spindle cell. No authentic cases of any variety of the osteogenic sarcomata have been recorded. *Gliomata.*—In no instance has any one of the classified varieties of the gliomata been encountered, and it is generally accepted that tumours of this type are very rare in animals.

Melanomata.—Under this group are included all growths which showed an abnormal collection of melanin pigmented cells. All were located in the skin and subcutaneous tissues. It is well known that such growths are of common occurrence in dogs and horses, particularly grey horses. The types encountered have varied

from the small pigmented nævi (single and multiple), warty and pedunculated in type with apparently benign histological characters, to large diffuse epidermal growths with metastases in the adjacent lymph glands. With the latter there is frequently a history of slow growth over a period of years in which trauma appears to play a part. Others apparently remain localized, but if interfered with may recur in a matter of months. An intensive study of this type in animals would perhaps help to elucidate many of the controversial points in connection with melanomata, e.g. the histogenesis of the melanoblast, the mode of origin and distribution of the pigment, and the relation of the simple pigmented nævi to malignant transformations. These cutaneous tumours offer an opportunity of studying pathological changes in the earliest stages from the simplest quiescent nævi to the gravest proliferation of the malignant forms. *Lympho-sarcomata.*—Two cases of this type have been observed in the dog. One was located in the mediastinum involving the mediastinal and bronchial lymph glands with secondary deposits in the lungs, the other was confined to the lower end of the ileum and mesenteric lymph glands with metastatic deposits in the kidneys. In both cases the regional invasive growth which is characteristic of this type in man was well demonstrated. The histology was that of the reticulum-cell or large-cell type. *Adenomata.*—These have been located in the mammary gland, intestine and liver. Anal adenoma in the dog is of frequent occurrence. *Fibrocystic adenoma.*—Two tumours of this type have been collected, being located in the testis of a dog. Both had a history of rapid growth but were apparently benign in nature. Periods of eighteen months and two years respectively have now elapsed since operation without there being any sign of regional involvement. Both were very large growths with much hæmorrhage and cyst formation. *Carcinomata.*—The *squamous* type was found in the skin, eyelid, mammary gland and clitoris and, in one instance, the heart of a cow, a very unusual site. No cases of the *basal-cell* type have been observed. One epithelial growth affecting the tonsils, with secondary deposits in the cervical lymph-glands and lungs, in a dog, closely resembled a type described by Ewing and others in man, and designated as *transitional-cell carcinoma*, or *lympho-epithelioma.* This type in man has a peculiar clinical course and is only located in the naso-pharynx. *Embryonal carcinoma* or *seminoma, of the testis.*—Five of these growths have been collected from the dog. This is a type of neoplasm which has not been recorded previously as occurring in this animal. The structure is identical with that of seminoma of man, namely, large polyhedral cells growing in cords and sheets, with a stroma richly infiltrated with lymphocytes. The tendency to alveolar formation has in the past led to its classification as alveolar sarcoma, but this is incorrect. In each of these cases there was a history of rapid growth. The epididymis was not involved and in no case has metastatic spread occurred after operation in periods up to two years. The affected animals were middle-aged or old dogs (5 to 11 years).

Dural endothelioma, psammoma, or meningoma.—Although only one specimen of this type has been collected, it is hardly a rare growth in the horse. The tumour, which probably develops quite slowly, is attached to and vascularized by the dura mater. It is never metastatic and only gives rise to symptoms as a result of the pressure it exerts. The commonest situation is in the falx.

Section of Anæsthetics.

[*February 5*, 1932.]

Hypnosis and Anæsthesia.

By Bernard Hollander, M.D.

Hypnotism for the prevention and alleviation of pain has been practised in some form or other throughout the ages. One form is familiar to us all. When a child who has hurt himself runs to its mother crying and she strokes the injured part, reassures the child, takes him up in her arms, and rocks or sings it to sleep, she is unknowingly exercising a hypnotic influence. The methods of priests and magicians, in ancient times, in healing the sick, appear to have been of a hypnotic kind; and the temple sleep of the ancient Greeks and Egyptians seems to have been of a similar nature. Celsus says that Asklepiades practised light friction as a means of inducing sleep in frenzy and insanity, and, what is more remarkable, he says that by too much friction there was danger of inducing lethargy. In all ages, and amongst all nations, a certain sanative efficacy has been ascribed to the touch of the human hand, to the placing of it upon a sick person, or rubbing with it any part of the body that may happen to have been exposed to injury. Healing by touch was practised by kings and professional healers in Western Europe until quite recent times.

Hypnotism, as practised at the present day, became popularly known at the end of the eighteenth century, when a Viennese physician, Friedrich Anton Mesmer (1734-1815), settled in Paris in 1768, and used what he believed to be "magnetic emanations" for the purpose of medical treatment. According to his view, the universe was filled with a magnetic fluid, more subtle than ether, which could be passed from magnetizer to patient, and he asserted that this "animal magnetism," as he called it, possessed great healing properties. Princes and nobles thronged to consult the "great magician," and in order to treat the crowd of patients that flocked to him, Mesmer gave up individual treatment, and invented his famous magnetic "tub," to enable him to give mass treatment. Public sentiment then turned against him, and the medical faculty attributed his success to the power of "imagination," and condemned him as an impostor. Whatever may be said against Mesmer's theory and the methods he employed, there can be no question that he genuinely applied them as a remedy for human suffering, and that often such a profound impression was produced upon the system of the patient as to effect the relief or cure of a certain order of malady. "Mesmerism," as the procedure was subsequently called after its originator, proved most effective in the production of anæsthesia for surgical operations, and has never been equalled in that respect.

One of the earliest operations performed in the magnetic sleep was in 1821 by

J. C. A. Récamier, Professor of Gynæcology in Paris. Similar ones were performed at other hospitals under the direction of Baron du Potet. One operation in particular created great sensation, namely that by M. Jules Cloquet, one of the most eminent surgeons of Paris in his time. Authentic details, of which I can only give a brief account, are preserved of it, for he reported it to the Academy of Medicine, April 16, 1829.

The patient, a woman, aged 64, suffered from cancer of the right breast, with considerable enlargement of the axillary glands, which caused her pain and loss of sleep, for which her physician, Dr. Chapelin, used to mesmerize her. It was he who proposed to M. Cloquet to operate while the patient was under mesmeric influence. The operation was arranged to take place on Sunday, April 12. An hour before the operation the patient went to Mass, as was her custom every Sunday. When M. Cloquet arrived at 10 in the morning he found the patient sitting undressed in an armchair in a mesmeric condition. Her physician supported her right arm; the left was allowed to hang down by her side. M. Pailleux, of the Hospital St. Louis, was in charge of the instruments. An incision was made from the axilla to the inside of the breast, both above and below; the tumour was then removed, and the engorged glands dissected out. The operation lasted twelve minutes.

Now comes the interesting part of the procedure:—

"During all this time the patient continued to converse tranquilly with the operator, and gave not the least sign of sensibility. No movement in the limbs or in the patient's features, no change in the respiration nor in the voice, and no changes in the pulse were observed." After the ligatures had been applied, the wound was united by adhesive plasters and dressed, and the patient was put to bed, and left in the mesmeric state for forty-eight hours. The first dressing was removed on the following Tuesday; and, during the cleaning and dressing of the wound, there was neither sensibility nor pain, and the pulse had its natural rhythm. After the dressing Dr. Chapelin woke the patient. The lady remained unconcerned, but when told she had been operated upon, and on seeing her children around her, she evinced considerable emotion, which was checked by immediately putting her to sleep again. (*Bull. de l'Acad. de Méd.*, Paris, Vol. II, p. 370.)

When Cloquet read this account to the Academy of Medicine, Lisfranc, also a celebrated surgeon, got up and declared that Cloquet must be either an impostor or a dupe. That opinion, however, did not deter others from operating under similar conditions. At another meeting of the Academy, on January 24, 1833, Dr. J. E. Oudet reported having extracted several teeth in the mesmeric trance; but he was received with the same incredulity as Cloquet.

Yet a change in the attitude of the French medical authorities seems to have set in a few years later, for on March 10, 1841, Dr. Talbot-Descourty, a surgeon-dentist, operated on a patient in the mesmeric state, in the presence of the Dean of the Paris Medical Faculty, and other eminent medical men.

In England, too, teeth extractions under mesmeric influence were frequent at that time; for instance, by Mr. Martin, a dentist of Portsmouth (1841), and by Mr. Rideaux, a medical practitioner of Southampton, who published five such cases in 1842. In 1844, Dr. John Ashburner, of 55, Wimpole Street, mesmerized a girl for Mr. Toms, dentist to the Middlesex Hospital, who extracted eight teeth at one sitting.

Mesmerism in England and Scotland had many sincere and learned advocates at this period, such as Gregory and Mayo, both Fellows of the Royal Society; Colquhoun, and others; but the leader of the mesmeric movement was Dr. John Elliotson (1791-1868), also a Fellow of the Royal Society, and one of the most distinguished physicians of his day. In addition to being Lecturer on Medicine in London University, he was also one of the founders of University College Hospital, President of the Royal Medical and Chirurgical Society, the first physician to practise

auscultation with the stethoscope, and one who enriched the British Pharmacopœia with several new drugs. A demonstration of mesmerism in 1829, by Chenevix, a Fellow of the Royal Society, first drew Elliotson's attention to the subject; but it was only after seeing Baron du Potet's exhibitions, in 1837, that he commenced his own researches. Every effort was made to induce Elliotson to abandon the subject, and, when he would not desist, invective and vituperation were heaped upon him, and the Council of University College Hospital was forced to prohibit the practice of mesmerism within the Hospital, with the result that in 1838 Elliotson resigned his position.

As a result of Elliotson's efforts, "Mesmeric Infirmaries" were established in Marylebone (36, Weymouth Street), Edinburgh, Dublin, and elsewhere, and many surgical operations painlessly performed upon mesmerized patients. In Exeter alone, two hundred operations were performed by Mr. Parker, a surgeon. A booklet giving an account of these operations was published in 1843 ("Numerous Cases of Surgical Operations, without Pain, in the Mesmeric State." By John Elliotson, M.D., F.R.S.). The most interesting case was one of amputation of a leg above the knee, performed in the District Hospital of Wellow, Nottinghamshire, and brought before the Royal Medical and Chirurgical Society of London in 1842. The mesmerizer was Mr. W. Topham, barrister of the Middle Temple; the surgeon, W. Squire Ward, of Wellow Hall.

> The patient, a labourer, 42 years of age, had an ulcerated knee-joint, which caused him the most excruciating agony and almost constant insomnia. He was mesmerized every day for ten days before the operation, which took place on October 1. The patient was soundly asleep while the flaps were cut. "The placid look of his countenance never changed for an instant; not a muscle was seen to twitch. To the end of the operation, including the sawing of the bone, securing the arteries, and applying the bandages, which occupied in all a period of upwards of twenty minutes, he lay like a statue." At one time a low moaning was heard, but it did not seem connected with the operation; for the surgeon, as a test, pricked the sciatic nerve forcibly with the points of a forceps, but without any increase in the moaning. Besides, the patient's countenance retained its placidity all the time. After the operation he "gradually and calmly" awoke. After looking round, he exclaimed, "I bless the Lord to find it is all over." He confessed that he had felt no pain at all. He was again mesmerized for the first dressing of the wound. "Within twenty-four hours after the operation he was singing. In three weeks he sat up for dinner, and had not a single bad symptom."

No sooner had the account of the case been read than up rose Mr. Coulson, a surgeon, who exclaimed, "The paper ought never to have been read, for the only point of interest is the non-expression of pain, and that is a common thing; no doubt the man had been trained to it." Other surgeons were of the same opinion. Marshall Hall, the famous physiologist, said that the case proved too much, or, rather, flatly contradicted itself, because, on the principle of reflex movements, "the sound leg ought to have contracted when the diseased one was cut." Sir Benjamin Brodie agreed with Marshall Hall. On the other hand, Mr. Wood, who had assisted in the operation, vouched for the accuracy of the whole account of it.

At the following meeting of the Society it was proposed by Dr. Copeland to expunge the record of the meeting, for he contended that "if the account of the man's experiencing no agony during the operation was true, the fact was unworthy of their consideration, because pain was a wise provision of nature, and patients ought to suffer pain while their surgeon was operating; they were all the better for it, and recovered better." The motion was seconded by Dr. Evans and agreed to.

Dr. Elliotson recorded in his Journal, *The Zoist*, altogether 16 amputations, 28 operations for tumours (some of them of enormous size), and 150 other surgical operations, besides numerous painless parturition cases, most of them witnessed by a number of medical men.

Most interesting are the records of the Hospital at Cherbourg of operations in the mesmeric state between 1845 and 1847, one being the removal of a portion of the lower jaw, and another, on Friday, June 4, 1847 (as reported in the *Journal de Cherbourg* and *Phare de la Manche*), when three patients had enlarged glands of the neck removed. Dr. Durand, a professor of philosophy in Paris, was the mesmerizer; Dr. Loyal, the operator; and Major Fleury, a military surgeon, the assistant. To this performance all the notabilities of Cherbourg must have been invited, for, in the list of those who signed the report, are the names of doctors, lawyers, clergymen, naval and military men, headmasters of schools, the mayor and other officials of the municipality, including the tax collector.

Elliotson's work was taken up in 1843 by James Esdaile (1808-1859), a graduate of Edinburgh, and Medical Officer of the East India Company. He was in charge of the native hospital at Hooghly in Bengal, and used mesmerism for the production of anæsthesia in thousands of surgical operations, including 300 capital ones. Amongst the latter were twenty amputations, three cataract operations, three for strangulated hernia, one lithotomy, one amputation of the penis, and about 200 for the removal of the enormous scrotal tumours due to elephantiasis, so common in India. They are recorded in his two publications: "Mesmerism in India," 1846; and "The Introduction of Mesmerism as an Anæsthetic and Curative Agent," 1852.

Let me quote the account given by Mr. Allan Webb, Professor of Anatomy of the Medical College of Calcutta, who went to see Esdaile's hospital on an operating day (October 15, 1846). He described how he saw the compound full of carriages, which had conveyed distinguished medical men and other eminent spectators who had travelled as much as fifty miles and "had braved the terrors of a burning sun to witness the extraordinary performance of a surgical operation free from any pain."

He found the patients, who were to be operated upon, in their beds in the square open courts. All were sleeping profoundly, while at the head of each bed stood the native mesmerizers. The first case was one of a large elephantoid tumour, which was afterwards shown to be fifty pounds in weight; next came one of cancer, "a dreadful-looking mass." Next came the paring down of a big ulcer in the leg of another patient. These patients never stirred, and slept like little children; "whereas, had they been sensible, this would have been torture, so excruciating, that the whole hospital would have rung with the shrieks of the patients." "I have seen many," Mr. Webb goes on to say, who had undergone these operations in the normal waking state, "with the cold sweat of agony bubbling up for hours afterwards; and a pulse so depressed, that it was hazardous to move them; whereas these patients were quite chatty and lively."

Dr. Stewart, another witness, reported that the whole scrotal operation took six minutes, including the ligatures to the spermatic arteries and some other vessels that spouted. For his own satisfaction he held the pulse of the patient the whole time; only towards the end it became intermittent. The patient was then fanned, and cold water dashed in his face, whereupon he woke, drank some water that was given him, and subsided again into perfect repose, when some more small vessels were tied. Dr. Stewart visited the patient the next day and found him looking well.

The Governor of Bengal appointed a commission to investigate the matter, which confirmed that, in respect of the six operations witnessed by its members, no pain was felt by the patients. Dr. Esdaile thereupon got charge of a small hospital in Calcutta for his specific purpose and exclusive use.

Dr. Esdaile claimed that he lost not a single case as a direct consequence of the operations, though some of them had been very severe. Only 5 per cent. of his patients had died, and these in consequence of lockjaw, cholera, and other intercurrent diseases. We must not forget that those were the old septic days before the advent of Lister.

Contemporary with Esdaile and Elliotson there was a Manchester surgeon, James Braid (1795-1860), who started mesmerism on a new career, and gave it a new name: hypnotism, from hypnosis = sleep. He had attended in 1841 the conversazione of Lafontaine, a professional mesmerist, and readily perceived that all the phenomena were not the result of trickery. He found that intense gazing upon an object, accompanied by concentration of mind, would displace the threshold of consciousness in a varying degree, depending upon the mental characteristics of the individual, and the circumstances surrounding him. The subconscious mind was thus brought into play. The first result was a condition resembling sleep—nervous sleep —neuro-hypnotism, or hypnotism for short ("Neurypnology," 1843).

Now, hypnosis is not sleep. True, we can use hypnosis to send a patient to sleep, but, unless contrary suggestions are made, he is wide awake, but his attention is concentrated on the operator, his voice and action, and he can carry on a conversation if so desired, as we have seen in Cloquet's case. In ordinary sleep, on the other hand, as soon as consciousness is lost, the subject loses connection with the outside world. Hypnosis is purely a psychical state, whereas natural sleep is dependent on changes in the circulation and chemistry of the brain. Under hypnotic suggestion people fall asleep without fatigue to help them, and may be made to sleep so deeply that even surgical operations on them do not wake them, but ordinary sleep needs to be helped on by fatigue and other physiological changes, and is often hindered by pain and pathological inhibitions.

Many of the patients who come to be hypnotized think they must fall into ordinary sleep. If they do so, it only hinders the process. I have had them on the couch "snoring"; that did not make them better subjects.

Braid was much struck by Esdaile's results, and had also used hypnosis in surgery, chiefly for teeth extraction. He practised the "gazing" method. Later, however, on finding that his experiments succeeded equally well with the blind, he dropped his ideas of hypnosis and developed the theory of "suggestion." He found that hypnotism acts subjectively and objectively, and that the expectant idea in the mind of the patient is the real agent. But just as "animal magnetism" and "hypnotism" involve a special theory, so does "suggestion," which, as I shall have occasion to explain further on, covers only a limited portion of the ground.

Braid offered to give a demonstration on hypnotism to the Medical Section of the British Association in Manchester, in 1842, but his offer was spurned. Altogether he aroused but little attention during his lifetime.

When we consider the hostile attitude of the medical profession towards the chief representatives of animal magnetism and hypnotism, and recollect that in France physicians who supported or practised mesmerism were struck off the Register at one time (after the unfavourable report of the Academy of Medicine), we cannot wonder that the subject on the death of its pioneers was relegated to the travelling showman.

Mesmerism as an anæsthetic might certainly have come into general use had it not happened that just as its merits were becoming known chloroform anæsthesia was introduced by Sir James Simpson in 1847. Orthodox medicine, committed to a denial of the efficacy of psychic healing, welcomed the new anæsthetic with acclamation.

The aseptic method of performing operations introduced by Lister has enabled surgery to make wonderful strides, but another factor in its success was the discovery of anæsthetics. What must have been the agonies of patients in pre-anæsthetic days, considering that, at the present day, people shudder at the thought of having the slightest incision made, without being rendered unconscious, or having a local anæsthetic applied!

I know of a whole list of surgeons, contemporaries of Elliotson, Esdaile, and Braid, who regarded mesmerism, or the hypnotic trance, as the accomplished fact

of anæsthesia and the long-hoped-for panacea whereby suffering humanity could pass unflinchingly through the ordeal of the surgeon's knife. In France, A. B. Richerand had tried it, and had pronounced in favour of its value, and other surgeons, less eminent, were willing to swallow the doubtful reputation of Anton Mesmer, and the risk of being struck off the register, so long as they could benefit their patients by employing methods which had been exploited by him.

On the other hand Alfred Velpeau, at that time the leading surgeon of France, like the British authorities I have mentioned, declared that the abolition of pain in surgery was a chimæra which it was no longer permissible to pursue. Magendie, the great physiologist, supported the view of Dr. Copeland, before mentioned, namely that pain was useful, and expressed doubt whether there was any real advantage in suppressing it by making patients insensible during an operation. He added that it was a trivial matter to suffer, and that a discovery the object of which was to prevent pain, was of little interest.

I have already explained how, after the death of their pioneers, mesmerism and hypnotism were relegated to the sphere of the travelling showman.

In 1879 such a travelling hypnotist, the renowned Hansen (by whom, by-the-by, I myself was hypnotized successfully), like the Abbé Faria, only just escaped imprisonment in Vienna for what were still held to be fraudulent practices. He was ordered to leave Austria, and went to Breslau, where he succeeded in convincing Heidenhain, Professor of Physiology, of the reality of hypnotism. Heidenhain investigated the phenomena seriously, and converted the German medical profession. Professors Krafft-Ebing, Obersteiner and Benedikt, of Vienna, Möbius and Wundt of Leipsic, Preyer of Jena, and Eulenburg of Berlin, to mention only a few medical celebrities who had seen Hansen's demonstrations, now took the subject up in earnest. Giving honour to whom honour is due, they recognized the merit of James Braid, and translated his work, which had been quite forgotten in England. In 1880 the British Medical Association invited Professor Preyer, the translator, to give a lecture on "Braidism," as hypnotism was then called. Ten years later the subject had made so much progress that, at the Birmingham meeting of the British Medical Association in 1890, a committee of physicians was appointed to test hypnotism psychologically, physiologically, and therapeutically. This Committee reported favourably, and, what is more interesting, recommended it warmly for therapeutic purposes, especially for the abolition of pain. Consequently, when, a few years later, at the request of Dr. (afterwards Sir) David Ferrier, I hypnotized some of his patients at King's College Hospital, there was no longer any opposition. Indeed, at Krafft-Ebing's Clinic in Vienna, where I subsequently worked for a year, hypnotism was a regular form of treatment for functional nervous and mental disorders.

In France, too, hypnotism was revived. Dr. Liébeault, of Nancy, was the first to take it up. He held that the key to all hypnotic phenomena lay in "suggestion," that is to say, he found that hypnotism was not even an instrument, let alone a curative principle, but merely a psychical condition in which another instrument—suggestion—could be more readily applied than in the normal waking condition. To put it in more concrete form, he found that a patient might be hypnotized every day for a month without advancing his recovery in the smallest degree, but that if, during hypnosis, it was suggested to him that recovery was about to take place, then, during the waking state, something within him proceeded to carry that suggestion into practical effect.

Liébeault remained in obscurity until his pupil, Dr. Bernheim, by the publication of his book on "Therapeutic Suggestion," made his theories popular. In a few years Bernheim had hypnotized about 10,000 patients, and was successful in 85 per cent. of his cases.

Meanwhile another large school had grown up, in Paris, under the guidance of

the famous Professor Charcot, who declared that the state of hypnosis was nothing but an artificial nervous condition, a neurosis akin to hysteria, the various manifestations of which could be aroused at the will of the hypnotizer, both by physical and psychical means. We know now that Charcot's view of hypnosis was largely influenced by the fact that his experiments were made chiefly upon female patients already in a highly nervous condition. When experiments are made upon other soil, the results are very different from those recorded by Charcot. Hence the Nancy school had steadily prospered, whereas the Charcot school had lost favour.

According to the Nancy school, all the manifestations of hypnosis depend entirely on suggestion, and, even when no words are spoken, there is still "unconscious," if not "conscious" suggestion. This is contrary to my own experience, and I hope to convince you of the fact that hypnotism is not all suggestion, for analgesia can be produced by purely physical means without the patient knowing what is expected of him. The word "suggestion" has been too generally adopted, as if it explained all mysteries. It is true, however, that hypnosis is a process which makes suggestions operate as they do at no other time, and that through them functions are affected which ordinarily elude the action of the waking will. I have frequently left boys, brought to me on account of bed-wetting and other troubles, and who were too young to know anything about hypnosis, alone in a room, gazing at a glass crystal, making no other remarks but that they must not look away and that I would come back in ten minutes, and, when I have returned, I have found them in the cataleptic state.

That bodily functions which are not under voluntary control can be influenced in that state is proved by the many cases of chronic amenorrhœa in young girls which I have treated, many of these patients having been cured in one sitting, merely by my telling them in hypnosis that their menses would appear that very night. It may be said that this clearly was suggestion, but no one has yet explained why a few words, quietly spoken, should produce such a powerful effect on an unconscious physiological process.

I hope to prove to you that the hypnotic state is largely a condition of more or less profound abstraction or absentmindedness. Psychologists would call it a state of dissociation.

I do not attempt to get the patient to sleep, but to produce a passive concentration—without conscious effort or strain—so that the subject becomes unaware of his surroundings, pays no voluntary attention to his environment, and, as I shall demonstrate to you, very soon forgets that he has a body and limbs. His senses are quiescent, except for the direction that one or other is given by the operator, so as to concentrate the attention. The state produced is identical with the state of reverie when a person is meditating deeply and does not notice his sensations. In reverie, as in hypnosis, there is such a concentration of mind that the external world is obliterated, and neither things, persons, nor events that are passing about one are taken notice of, and what is of more importance, the body is ignored, and any discomfort or pain that may have pre-existed is now negligible and not felt. This state is also akin to that of a man in ecstasy over his work, who, for the time being, is relieved from all his bodily woes, and may even forget his earthly needs. Moreover, he may feel no pain, as happened to Marini, when writing his "Adone," when he did not notice a serious burn of his foot.

Many of the ecstatics and mystics of the Middle Ages, who were accused of being in league with the devil, must have possessed the gift of self-hypnosis, for when they were made to walk on burning coal or red-hot iron as a proof of their innocence, they did so without apparent pain or damage. Some could hold a hand over a blazing fire without flinching.

Ecstasy is merely a superlative degree of attention. It is a state in which all sensations and thoughts are suspended, except the one which forms the subject of

contemplation; the same as in hypnosis. In religious ecstasy the self-absorption may be aided by fixing the gaze upon some holy figure, just as we get the subject for hypnosis concentrated by looking at some object, and—as I shall explain in a moment—directing the eyes upwards. After all, what do we mean by saying that a man seems as if "hypnotized," but that his whole interest is so concentrated on one point that he neglects everything outside himself and every sensation? The visions of ecstatics may become realities to their minds, as when they see angels and other supernatural beings, not visible to anyone else, and the same thing happens as regards the visions suggested to the persons in a state of hypnosis.

That in certain states of mind, pain is not conveyed to the brain—that it does not become a conscious reality is proved, also, by the fact that we sometimes see madmen commit horrible mutilations with very blunt instruments, without exhibiting any sign of feeling.

Another familiar example of the effects of concentration may be observed, when a man in the street is made to look up to the sky, in expectation of seeing some strange object, and he gazes so intently, that he does not feel the hand of the pick-pocket extracting his watch. It is a well-known trick of thieves to get an accomplice to knock against a man suddenly, so that he becomes indignant; during his emotional pre-occupation he does not notice a robbery being effected and only becomes aware of his loss later.

The late Mr. Alfred Wallis, a well-known dentist and member of several committees of the British Medical Association, was so well acquainted with my methods, through the patients I brought him, that one day he practised them on me without my knowledge. I was seated in the dental chair, and had the gag fixed in my mouth, and saw the anæsthetist holding the face-piece a little distance away from me, while entertaining me on a topic of great interest (evidently instructed by Mr. Wallis), so that I became absorbed in his talk, until he made me laugh, when I remarked that it was time to get ready, and was thereupon shown the tooth extracted by Mr. Wallis, without my having been conscious of it. I complimented the anæsthetist on his cleverness. I must mention in this connection that I knew that Mr. Wallis, in his hospital practice, often directed his assistants to apply gas, whereupon the face-piece was applied, but no anæsthetic was given, yet the patients felt no pain whatever.

Some of those who hold that hypnotism is all "suggestion," have declared that the effect of chloroform is occasionally due to suggestion, inasmuch as frequently the patient is asleep after a few breaths. I am not in a position to decide the question, but in connection with the procedure of Mr. Wallis, just mentioned, it may interest you to know that Dr. Hallauer (*Berliner klinische Wochenschrift*, 1908), a specialist for diseases of women, has performed painless operations in the same manner. He proceeded as follows: All the preparations for an anæsthetic were gone through as usual. Once on the operating table, he applied from ten to fifteen drops of chloroform to the mask in full view of the patient, and told her to breathe quite quietly. When there were any movements, struggling or coughing, he removed the mask for a few minutes, and carefully placed it again over the face as soon as this had subsided. Then he added from time to time two or three drops of chloroform, mentioning the fact aloud as he did so, and stroking the forehead or hair, always in one direction. He explained to the patient that she was getting sleepy, and that sensation was gradually disappearing. The suggestion that complete unconsciousness was setting in came next, after actual drowsiness had begun. Hallauer's method did not require more than from twenty to forty drops of chloroform for any one operation. As soon as the operation was over, he told the patient to wake up, which she did without experiencing any ill after-effects. Even patients who had been a little restless during the operation declared that they had felt

nothing. One of the chief advantages of Hallauer's methods is, in my opinion, that if it fails, it is easy to continue the administration of chloroform in the usual manner. The same can be done with any form of hypnosis, for the purpose of anæsthesia, so that it can be definitely stated that in no case can harm come by it to a patient.

Local insensibility to pain, for minor operations on the throat, has been induced by Barth, by a similar method of suggestion, namely, by persuading the patient that the solution of common salt with which his throat was being painted was cocaine, and, therefore, rendered the mucous membrane insensitive.

After these preliminaries you will want to know how I proceed to induce the hypnotic state. I shall give you first a general description, and then show you how the methods can be applied to producing analgesia and anæsthesia.

I get a large number of patients sent to me, generally as a last resource, for the relief of neuralgia, neuritis, migraine, and insomnia due to pain, but the majority of my patients are addicted to drink, drugs, or abnormal sex habits, or suffering from morbid fears and obsessions, stammering and muscular tremors, or are mental cases on the borderland. I therefore follow a definite plan with all of them, that is to say, I try, first of all, to acquaint myself with the individuality of the patient, encouraging him to talk about himself, his history, his illness, and so forth. This enables me to adapt my voice and manner to the mentality of the patient and establish a cord of sympathy, which not only facilitates subsequent hypnosis, but enables me to make, when he is in that state, the right suggestion, and such appeal to his intellect and emotions as will be most readily accepted by him.

No anæsthetist should neglect a short talk with his patient in order to get in touch with him, for everyone feels anxiety when undergoing an operation, even when it is not so serious as to involve the possibility of a fatal issue, and anxiety is an emotion which has a disturbing effect. Therefore, a judicious and kind word spoken may act beneficially on the patient. With this you are all familiar, but there is something else you can do by suggestion. Such a remark, after examining the patient, as "the heart is wonderfully sound," or "what a splendid pulse, notwithstanding the long illness!" or similar observations made to the surgeon or assistant within hearing of the patient, is taken much notice of by him, and, so to say, "tunes" the mind for the operation. Let us not forget that the patient on the operating table is often already half-hypnotized, and the final words are pondered on, and may persist, even when he awakens and is in a state of exhaustion, or nearly dying. I have often had to prepare patients who would not give consent to a necessary operation. One suffering from cancer of the pylorus told me how happy he was on awaking from the anæsthesia, feeling no discomfort whatever.

The patient may be seated in a lounge chair, but generally I get him to lie down on a couch, with the body relaxed, and the mind passive and emotionally calm. Practically all the failures, when they occur, are due either to the nervous state of the patient, or his active thoughts. Nervousness can be remedied easily, but the patient pursuing his own thoughts is often almost hopeless. Of this description is the man who keeps on thinking: "I wonder what that fellow is going to do next," or "Ten minutes are gone and I am not asleep yet," or, worse still, the man, generally a drunkard, who wants to get well, as if by a miracle, at one sitting, able to pass every public-house on his way home without taking a single drink, and having done nothing to become really reformed.

The patient lying comfortably on the couch, I then let him look at some object, generally the globe of a lamp, quite quietly, without strain, but keeping his eyes on it, so that he notices nothing else. The effect, after a time, is that he sees only a mass of light in front of him, and can no longer distinguish the globe. A tired feeling of the eyes becomes perceptible to the operator, and it will be seen that the patient then either closes his eyes from fatigue, or turns them upwards, so

that the pupil disappears from view, or he may merely start blinking with his eyes In any case he is told now to close them.

When in my own treatment room, I usually set an electric motor going at this stage and get the patient concentrated on the monotonous noise of it, so as to tire the auditory nerve as well as the optic, and to enable him to ignore other sounds which might wake him. But this procedure is not essential. I use it because I have found that very few persons can keep staring at a light and listening to a monotonous sound without getting drowsy.

We have now got the patient in the somnolent state by the process of gazing. But we can also get him into that state by the Nancy method of talking to him, using persuasion and suggestion, describing to him the successive stages of sleep: "Your eyes are getting heavy; you are getting tired; you can hardly keep your eyes open; they are closing now," and so on. I prefer trusting as much as possible to physical effects, because I have found that talking to the patient rouses the critical faculty.

Having got the patient to the therapeutic stage, the followers of the Nancy school, if they want to influence the patient's power of movement or his sensations, will now say to him: "You find it difficult to move your limbs, still more difficult, now you cannot move them at all"; and again, "Your pain is easier; it is getting less and less; now it has ceased altogether." This method is excellent, so long as we succeed with it, but woe unto us if we fail, for the patient will become convinced that hypnosis is no good to him, and that will be the end of it. Therefore, again, I trust more to physical methods which save me talking and will produce the identical effects automatically.

The subject, lying quietly on the couch with his eyes closed, is first of all asked to breathe deeply and regularly: "1 - 2, 1 - 2." I have added occasionally: "Think you are inhaling an anæsthetic, and getting less and less conscious." This, of course, is suggestion, and is not essential. It is held by the Yogis in India that such regulated breathing alone is apt to induce anæsthesia, and this, according to Moll, has been confirmed by Bonvill and by Hewson.

I then ask the patient to keep his eyelids closed and to turn his eyes to the centre of the forehead, i.e., upwards and inwards. This is the position the eyes are in at the moment when we go to sleep at night, and causes the mind to become a blank. If the patient is now told to keep looking to the forehead, and to attempt to lift his eyelids, he cannot do so, and will succeed only in raising his eyebrows. This manœuvre carries conviction to the patient that some powerful influence is beginning to assert itself. Indeed, a large number of patients find it difficult now to "think" at all—especially if the hypnotizer's thumb be pressed against the middle of the forehead of the patient—and cannot even make the effort to remember their name or address. Hansen, when he hypnotized me, held his thumb to my forehead, and made me look up. When he asked my name, I have still a vivid recollection that I could not make the effort to think, and that, when he suggested it was "Brown," it gave me relief to assent to it, though I had a feeling that it could not be. Of course, if the patient were to turn his eyes down, he could at once remember, but a good subject will not disobey his hypnotist.

What this turning-up of the eyes actually does is to increase the concentration of the attention, focussing it on the hypnotist, his words and actions. David Ferrier found, long ago ("The Functions of the Brain," 1876), that the movements of the eyes were concerned with the power of attention, and when he experimentally paralysed the muscles of the eyes (by the destruction of the frontal lobes) in monkeys "the animals presented an aspect of uninterest and stupidity," attention was lost, and the animals could no longer think, or take notice of anything. Professor Elliot Smith, in his Montgomery Lecture, January, 1922, has further explained the importance of the movement of the eyes "as a most profound stimulus to almost every

part of the cerebral cortex," and that "the fixation of vision represents the germ of the powers of attention, and mental concentration in general."

This turning up of the eyes, I have found, has another powerful effect which is of the greatest interest. Sensations from the body appear now not to reach the brain, or, more correctly speaking, do not seem to become conscious. Indeed, there is a feeling as if the body had become detached from the mind, as patients have confessed to me subsequently. Owing to the loss of sensation, the body and limbs are felt only as heavy masses, and there is immobility, or, at least, a loss of power to move them. In a good subject we may now raise an arm at right angles to the body, and it will remain in that position to the end of the sitting, whereas, in the normal state, it would soon droop. This insensitiveness of the body may go so far that the subject will not be conscious of the prick of a needle, or a sharp pinch. If the patient has suffered from pain, he does not feel it now, and can be told it has gone for ever.

Public performers of hypnotism often thrust long needles into the muscles of hypnotized subjects. I have witnessed several such performances in public theatres, and one given for my own private benefit. In addition to other experiments, the operator pierced a hand of the subject rather deeply with a darning needle, and had a difficulty in getting it out again. The hand did not bleed, but it became red. When I expressed my anxiety as to the consequences, the operator just made a few passes over the hand and the redness disappeared. He then woke the subject. I at once asked him whether he felt any pain, but he said "No," and on being told by me that he had been stabbed with a long pin, he searched for the injury all over his body, and when I directed his attention to the hand, he turned both hands over and told me he could see and feel nothing whatever of having been hurt anywhere.

One fact must not be overlooked, that is, that sick people are more suggestible, and more readily hypnotized than ordinary folks. I have never induced hypnosis for a surgical operation, but I have frequently been sent for by surgeons when patients continued to suffer pain after operations, where the usual analgesic and hypnotic remedies had no effect.

Once I was asked by a surgeon to go to a nursing home in Hampstead, where a young woman, dying from cancer of the uterus, suffered great pain which caused her to be sleepless. Within a few minutes the patient was asleep, and, in a few minutes more, wide awake with spasmodic pain. I persevered for an hour, at the end of which she seemed more peaceful. I heard no more for a week, when I received a letter thanking me for what I had done and stating that the patient had slept free from pain every night since my visit.

Another case, also in a Hampstead nursing home, was that of a medical student who had been run over by a motor lorry and had sustained a smashed ankle. After the operation he suffered agony. The joint was resected, but no cause for the pain could be found. Then medicinal remedies were tried in vain, until the physician in attendance asked the operating surgeon to permit a trial of hypnosis. Again I had some difficulty, this time because of the resistance of the patient, but in the end I succeeded, as was acknowledged by the patient's doctor, who called a few days later.

Another case was that of a boy on whom a mastoid operation had been performed. He suffered great pain after the operation and said that it hurt him to eat. Again the wound was opened up, but nothing disturbing was found. The surgeon then recommended a course of ionization treatment, but that too was in vain. Then somebody suggested a trial of hypnosis. This time I formed such a favourable opinion of the case in question, that I ordered a wonderful tea, with all sorts of delicacies that a boy loves, to be ready after the treatment. Actually, the pain was gone after the first attempt at hypnosis. I asked the patient whether he thought he could eat now. He replied that he would try, so I invited him to have tea, when

he ate of all the things provided for him with great relish. That boy is now a distinguished Don at Oxford, and I know positively that there has been no return of the pain.

Another case of a different character was in a Wimpole Street nursing home, where a young woman, just returned from India, suffered from insomnia and chronic vomiting, the origin of which puzzled her physicians. After antitoxins and other remedies had been tried in vain for weeks, one of the consultants thought the cause might be mental and sent for me. This was another example of those spontaneous cures, which, even though they occur rarely, are possible only in hypnosis. The patient slept well already the first night, and the vomiting ceased on the second day; and the sensation in the nursing home was so great that even men with broken legs asked for the doctor who cured Miss A.

Other extraordinary results to be achieved by hypnotism have been shown by me at private demonstrations to neurologists, alienists, and other members of the medical profession, and published in my books on "Hypnotism and Suggestion" (1910), and "Hypnosis and Self-Hypnosis" (1928).

I have already mentioned some of the early mesmerists who extracted teeth from patients in a trance. In more modern times there was Dr. J. Milne Bramwell, who, in 1899, created a sensation in medical circles by having hypnotized a number of patients for Mr. Arthur Turner, a dentist of Leeds. Then there were dentists who hypnotized their own patients: for instance Andrieu, of the Charité Hospital; Hivert and Meiroud of Paris; Klemich of Bromberg; Sandberg of Skofke in Sweden (1892), and also Glogau of Frankfort, who recommended its general use in dentistry. Amongst English dentists who practised hypnosis were Charles Childs of Bungay; Nicholls of Burton Street, Mayfair; Nasmyth, Surgeon-Dentist to Queen Victoria; Carstairs of Sheffield, and Dr. Owens of Stourbridge.

Hypnotism has also been frequently applied for the pains of confinement by Dr. Wetterstrand of Stockholm (1887); Mesnet (1887); Secheyron of Paris (1888); Auvard (1887); Thomas of Toulon; Varnier (1887); Voigt of Leipsic; de Jong; Tatzel of Essen (1894); Dr. Kingsbury of Blackpool (1890); Dr. von Schrenck-Notzing of Munich (1891); Grandchamps (1889); Dr. Luys of Paris; Prof. Cajal of Barcelona; le Menant des Chenais of Paris; Delboeuf and Fraipont of the Lying-in-Hospital at Liége. Dr. C. Pritzl published several cases: one, that of a primipara, was fully described in the *Wiener klinische Wochenschrift*, November 7, 1885. Dr. Lafontaine published two cases (quoted by Gilles de la Tourette, "Hypnotism," 1889). Dr. Dobrovolsky, a Swiss obstetrician, reported eight confinements in the state of hypnosis. Altogether quite a respectable number of such cases have been recorded. In all, the contractions of the womb were regular and strong, and perceived, but no pain was felt.

During this revival of hypnotism, hypnotic anæsthesia in surgery was again applied by, among others Professor Forel of Zürich; Tillaux and le Fort, two Paris surgeons; Voigt of Leipsic; Grossmann of Berlin; Howard, Professor of Surgery in Baltimore; Wood and Toll of Minneapolis, and Professor Stark of Heidelberg. Dr. Bourdon of Meru in France operated for fibroid tumour of the uterus (1898), and Dr. Schmeltz of Nice removed a sarcomatous tumour of the breast and a sarcoma of the left testicle in hypnosis (*Revue de l'Hypnotisme*, Vol. IX, 1897). In 1890, Haab, the celebrated eye surgeon of Zürich, used hypnosis in the operation for cataract. Lamphear, Aldrich and others, are mentioned by Moll as having used hypnosis for amputations, and Hulst for gastrostomy.

The doctors who have practised hypnosis for anæsthesia claimed the following advantages for it:—

(1) No abstinence from food or other preparation is necessary.
(2) Nervous apprehension can be removed by appropriate suggestion.
(3) Hypnotism is pleasant and absolutely free from danger.

(4) It can be maintained indefinitely, and terminated immediately at will.
(5) The patient can be placed in any position without risk.
(6) There is no tendency to sickness, during or after the operation.
(7) Pain after operation, or during subsequent dressings, can be entirely prevented.
(8) The rapidity of the healing process, possibly as the result of the absence of pain, is frequently very marked.

To this I would add a few further observations in order to dispel misgivings.

Some people fear that a patient might not be awakened readily. Let me assure you that nothing is easier. I tell my patients that in fifty seconds (or any other time selected) they will be wide awake, and, exactly to time, they will get up as well as if nothing had happened. Another method of mine is to tell the patient: "You will now count to 5 (or any other number) when you will awake, perfectly conscious, and well." And he does so.

Others want to know how, supposing the operator has said to a patient in hypnosis, "You will keep perfectly free from pain," or "You will touch no more drink," he can be sure that this will come true. That also is simple. In perfect hypnosis the patient has no recollection of what has taken place, until he again becomes hypnotized. I am therefore able to add that, when the patient wakes, he will, after a few minutes, complain to me of stiffness in his arm or leg, or his mouth will be rather dry, and he will ask me for a drink of water. It is beautiful to observe the expression of the patient on waking, till he summons courage to speak to me about his discomforts. But if he does so, unconscious of the fact that it was on my suggestion, I know that I can trust him to carry out the other more important directions without fail.

It is said that a person with heart disease should not be hypnotized. Frankly, the first time I had a heart case I was nervous about it. Now I no longer mind treating these cases. One of the worst I ever had was in a girl suffering from chorea, who had valvular disease of the heart following on rheumatic fever.[1] When I got her on the couch her pulse was 140. I quietened her with words and passes, and within a few minutes her pulse was practically normal. As regards the chorea, she was another of those spontaneously recovering patients, which you can find only in the application of hypnosis. But I have no theory to offer you why those patients who make good hypnotic subjects recover so marvellously. I leave the inquiry to the research student of the coming generation.

With regard to anæsthesia, there can be no doubt whatever that the anæsthetics of the present day are much surer in their action than hypnosis. No one in his senses would dream of replacing them. But there are cases of dangerous organic defects in which hypnotism might be preferable, or both anæsthetics and hypnotism might be tried together. It is well to remember, also, that persons who have been hypnotized before, will ever afterwards make good subjects, and can be used for the induction of anæsthesia should a surgical operation ever become necessary.

I have given in this paper a collection of facts and experiences which, I hope, will convince you that when we label certain phenomena as "mesmeric," "hypnotic," or "due to suggestion," we are really only playing with words. The fundamental process by which mind influences mind, and the mind influences bodily states and functions, is still wrapped in mystery. No patient, however extraordinary, and no number of patients that I might bring before you, would carry such conviction as you would gain by your own experiments on suitable subjects. It is high time that systematic research were undertaken into these phenomena, in place of the scattered evidence of a few individual experts.

Dr. A. W. MATTHEW said that he had had two cases in which the administration of an anæsthetic had been associated with hypnosis. The amount of anæsthetic used in both cases was negligible—one drachm of 50% C.E. mixture in one case and under two drachms

[1] See *Medical Press*, 1912.

of a similar mixture in the other case. The hypnotic state was produced accidentally in both cases and occurred at the moment that the patients were told to "go to sleep." The condition was characterized by an ashen grey colour, complete relaxation, absence of all reflexes, a dilated pupil, regular pulse and respiration, and absence of hæmorrhage. The administration of oxygen had no effect on the colour of the patients. The return of consciousness was sudden and complete and was accompanied by normal oozing from the raw surfaces. The operations performed were the enucleation of tonsils, and a submucous resection of the septum in an unprepared nose.

The absence of hæmorrhage was, perhaps, the most alarming feature of this condition, and it suggests that hypnosis is intimately associated with the vasomotor system.

Section of Psychiatry.

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The Experience of Time in Mental Disorder.

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The idea of evolution in time has been the main influence in biology for many decades; and in philosophy the universe is a four-dimensional continuum of which time is an essential aspect. In narrower fields it is the same. That disease is a matter of time relations was the opinion of Clifford Allbutt. Hughlings Jackson and von Monakow have insisted on the importance of a time-factor in all the normal and pathological changes of the nervous system; while investigations such as those of the physiologists into chronaxy are another recognition of its significance. It would be strange if psychology and psychopathology, always responsive to current modes of thought, should here be lacking. Nor are they, but one can still comfortably read through all the available literature, written by a handful of German and French psychiatrists, on time-phenomena in mental disorder. There are two aspects of this subject. The first is the time factor as a determinant of the duration of illness: a factor too often ignored by psychotherapists of the thaumaturgic kind—this I do not propose to consider. The second is of time as a modality of personal experience, an immediate or derivative datum of consciousness. Gross disorders of this occur in organic psychoses, where disorientation in time is a familiar feature, but it is also found in the "functional" psychoses, and it is with the latter that I am here concerned.

An extract from the case record of one of my patients will illustrate the matter.

"Everything seems very much longer. I should have said it was afternoon, though they say it is midday. They always tell me it is earlier than I think . . . . . and it looks as if I'm wrong and I can't help feeling I'm right . . . . . I cannot see any end to anything, any end to the world . . . . . I don't know whether I shall die quickly within what we call time and then have no more feeling . . . . . Time has a limit, but I don't know what limit . . . . . Time certainly passes, there is progression but not in the ordinary way now; there seem to be long pauses and then it goes on in the ordinary way . . . . . I've felt twice that time is standing still . . . . . I suppose I judge my time by the way I feel where the sun is; I noticed it very suddenly changes as though at the will of someone . . . . . I think no one will ever die."

This is fairly typical of one kind of time disorder: and one hears such accounts not only from the highly educated or philosophically minded but also from unintelligent, illiterate or matter-of-fact patients. In such a patient as the above there are unquestionable changes in the sense of time. To examine these one must have some notion of the psychology of time, i.e., of the mental happenings which permit us to recognize the present, judge the duration and passage of time, and bring it into relation with other contents of consciousness.

Time is a word of several meanings. There is objective physical time, clock time, calendar time, and there is personal time, experiential time, in short, psychological time. The former, which may be called world time, is the arbitrary standard, a fictional construction. The latter is time that we live in. They are usually more or less in agreement, more especially where no disturbing factors enter, as in sleep,

hypnosis, and in certain waking states. But psychologically they are quite separate. Time-consciousness is a matter of immediate experience: it is an aspect of all conscious activity: it is essential to all reality: whatever we call it, *durée vécue* or what not, it is inescapable in every reality-experience: we live in time. J. S. Haldane puts it summarily: "When we perceive things they are perceived as related to a past and future, and as related spatially to one another . . . . . Past, future, and contemporary present are thus given in each experience . . . . . our perceptions express our interest. In other words our perceived world is no mere picture independent of our presence in it, but the embodiment of our personal interest which reaches back over the past and forward to the future, so that past and future are represented in the present. Each of our perceptions, and each of our actions, embodies learning from experience, and therefore both retrospect and foresight; . . . . . ; when we see evidence of this we interpret the behaviour as being conscious. Unity extending over events in time as well as space is expressed in conscious behaviour."

Time is by no means an attribute of sensation, though probably of perception. Those who consider time from a genetic point of view hold that the elementary primitive phenomenon was duration: this, in its dependent relation to conduct, was developed into the idea of stability which was extended and objectivated so as to become a feature of the environment, otherwise so changeable and manifold. Beginning and ending are indispensable integral parts of duration; rhythm, so important in biology and art, is a mixture of stability and change. The conception of the present is a later development; with it comes the conception of the absent, to which we adapt ourselves by expectation. Memory has developed in order to integrate past experience with the living present. The present is set apart as a particular point, "now," in relation to which the succession of happenings can be put in order as past and future. Otherwise one has pure and simple succession, as in children who mix up the order of events. Thus the present has a constant relation to action and striving. This setting up of a notion of the present is possibly a complex, not a primitive, mental process; through it meaning is given to the present. In *déjà vu* there is a brief inability to actualize the present which in consequence is projected into the past. The present and the past are not uniform: there is an immediate past, a recent, a remote, and an indefinite past (and future), graded according to their closeness to the present and intimacy with it, as it were, and according to their explicit emotional character. The immediate past is not a matter of recollection: it is a progressive reality, almost continuous with the present, but shading off into the more remote, the mass of forgotten but available past from which can be evoked clearer recollections by bringing them up into the immediate past. Our conception of the past is like our consciousness in general: the totality of both consists in a global feeling of which only certain modalities attain to clear presentation.

With the penetration of the notion of time by the idea of reality the essential features of the process are arrived at. Instead of giving the same reality to past, present and future, the present is now regarded as real, past and future as unreal: diminishing degrees of reality attach to the immediate, near and remote past, as to the immediate near and remote future, but the indefinite past and future are almost entirely unreal, and such remoteness as is implicit in eternity is for us ordinarily an abstract fiction.

As a form of philosophical organization of time, the general and universal idea of time has developed, like the corresponding idea of space—something independent of ourselves; something external, unitary and abstract; a time which passes and destroys. A scientific mode of organization of time has led to the introduction of objective measures as well as to a determinist conception of time, as the conservative, yet always dynamic, transmitter of past forms of energy into forms yet to come.

The objective measures of time are familiar divisions of "world time," in which the present is a travelling point—not a brief brightest area in a scale of brightness shading off as it recedes at either end: in it the past disappears. Personal time is often brought into relation with world time. We have a consciousness that time is passing quickly or slowly in an absolute sense: also we may conclude that it is passing quickly or slowly in relation to world time; the two are commonly associated but by no means identical. In boredom, for example, time is said to pass slowly, because it is not possible to give it the content one would choose; hence, secondarily, comes distressing attention to the passage of time. Interesting changing occupation gives one the time consciousness "how fast it goes," though after it one has the impression of a long day, full of incident: a dull tiresome day seems, to retrospection, short. But this time consciousness is different from "time sense," which enables us to guess at the duration of an interval in terms of world time. Some would limit this as a simple perceptual process to very brief intervals (one and a half to two seconds), and regard the appreciation of longer intervals as a matter of judgment: they talk further of memory images, and of intervals with and without stimuli, but these notions and experiments and this element in the process, if it be one, need not detain us. What is the basis of this quantitative awareness of time, whether it be of long intervals or short ones? Several explanations are current among experimental psychologists: some referring to feelings of tension, others to contents of sensation, and so on; generally they mistake the factors which hinder it or alter it for those which are its basis. Others speak of "psycho-energetic" processes which appear in consciousness as a feeling of activity; thus they connect the quantitative appreciation of time with the psychic concomitants of action. But this alone is not helpful. One must consider, I submit, two elements: (1) an immediate awareness of the magnitude of duration or of speed of succession; and (2), an ability to recall similar former experiences for comparison. I do not suggest that the latter are simple memory images or engrams, plucked out of their pigeon-holes for the purpose, as schematizers would have it, but as representing particular aspects of memory, to which I have already alluded. As to the former, it is carried out best in sleep and allied states; in waking hours the less of affective interest and direct attention to it, the better it is performed. The psycho-analysts say it is a function of the pre-conscious part of the psyche. It is, I would suggest, a particular manifestation of the general time sense of the integrated organism, also manifested in such periodic activities as cardiac and respiratory function, sexual activities, e.g., menstruation, the alternation of metabolic changes associated with sleep and waking. Various writers pick out particular activities of this kind and hold them responsible for the special capacity: it is better to consider it as deeply rooted in biological changes of the whole organism. I would refer, in passing, to the work of Pawlow's school in regard to the conditioned time-reflex. It is not appropriate to discuss the relation of rhythm to time in further detail here: one psycho-analyst of repute thinks that time feeling is unconscious and depends on unconscious rhythm being an endopsychic perception of fluctuation of libido-distribution.

Only with some general notions, such as the above summary of views on the psychology of time, can one set about an analysis of the disorders of time processes that we meet in our patients.

What are these disorders? First let us consider depersonalization and the sense of unreality. The following is a description of his time disorder by a young man with a striking clinical picture of this sort:—

"As I went about everything seemed unreal. There is no break of time. I'm passing through time, there is no day and no night. There's nothing divided between my getting up and coming here and going back It's all joined into one. I have no definite recollection of going to Australia, though I'm only just back. . . . There's something gone from

me—the spirit: I have no life. . . . I'm dead already. I can't die. The mind is alive. You can't kill that. You could hurt the physical body, but if you were to shoot me, I would drop to the ground. But 'I' would still be sitting here, it must go on and on . . . it's a trance, a dreadful dream. My mind is there for eternity, nothing can alter it. . . . You can't imagine yourself different from the present, can you? Well, I'm that, accentuated a hundredfold."

Here is another, also from a young man:—

"Everything seems just a painted picture, deathly quiet. . . . I want to get something back to my mind that seems to have gone, to let me see the present and the future . . . . everything seems ages ago. Even getting up in the morning, I feel I've forgotten it; it seems ages ago. And yet time seems to go faster. If anybody had asked me at this time (12.30 p.m.) I'd have thought it was only half an hour since I got up. It simply means that time is going quicker than I realized. . . . Time does drag naturally, having nothing to do. I can't visualize; I can't picture anything at all. Everything comes new to me and fresh. . . . The future to me is remote. I feel hopeless. . . . Before, I could look to the future, but I can't now. There's something won't let me (actually continually worrying about the future). Time just doesn't appeal to me. I look up and see it is twelve o'clock, and think, 'What's twelve o'clock?' . . . I've got no idea of time or what the day is or anything, unless somebody tells me. I mean, I couldn't tell you how long I've been sitting here; an hour seems like a minute. Time doesn't seem to go at all; the whole day seems exactly the same. If I look at the clock it doesn't mean anything to me at all. . . . I couldn't give you the remotest idea now what the time is [asked, said it was twelve o'clock, actually 11.50]. It's difficult to think of the past at all . . . everything seems to have gone out of my head."

These are not rarities or exceptions. They are typical of numerous accounts that I have collected. They illustrate many of the outstanding features of the disorder: the inability to evoke the past readily or clearly, to distinguish the present from the past and the future ("I think about them all three at once, past, present and future," said another patient), to seize the present, to look into the future or to anticipate a future for oneself; there is paradoxically the increased quickness with which time passes, though it seems also to drag along; the seeming remoteness of the recent past; the unconfirmed feeling of inability to judge length of time.

The point-by-point application to these phenomena of the considerations presented earlier in this paper is, no doubt, sufficiently apparent. I will, however, put forward some more general considerations. The influence of MacCurdy, or an itch for causality-explanations in terms of comprehensible *einfühlbare* psychic processes, has led to this syndrome being regarded as a sequel or "manifestation of loss of interest which is in turn related to the loss of energy and stimulus susceptibility." I have elsewhere put forward evidence against this view. I would regard the syndrome as indicative of the disturbed state of consciousness in which the immediate data of perception—an active, not a passive process—are so changed as to have lost their primary attribute of reality. There is a change in space- and time-consciousness which may be expressed in various forms: simply, as in the above instances, or less clearly in general feelings of a change in the environment, or of complex rearrangements of space and time relations. In short, clinically one finds the syndrome of unreality and depersonalization, in various guises but none the less unmistakable, in many kinds of mental disorder—in manic and depressive states, in schizophrenia, in obsessive-ruminative tension states, encephalitis lethargica, toxic confusional and delirious forms, epilepsy and hysterical dissociations. That is easily demonstrable clinically; without going into all its implications I suggest that in all mental disorder there may be more or less disturbance of consciousness *in this sense*, and that this disorder of consciousness, in affective as well as other forms of mental disorder, may manifest itself only in a trifling time and reality disorder, hardly distinguishable from the variations we are aware of in ourselves (who are presumably normal) up through a growing obviousness to the grossest forms of disorientation and distortion.

I have not referred to the disturbance of space appreciation in this syndrome, but it is of the same order.

The clearest analogue to depersonalization and unreality in ourselves, if indeed it is not the same thing, is the *déjà vu* or *déjà raconté* phenomenon. I think immediate premonitions also must be included here; in this connection I would recall what was said about expectation in the earlier part of the paper. I have been impressed in reading a large number of records of the unreality syndrome to find how regularly it is associated with a feeling of imminent disaster. I cannot here consider its relation to anxiety, but I mention it, and refer also to the statement which patients make that "there is no future": this invariably refers only to the personal future, sometimes only to a pleasant or tolerable future: "I feel nothing will change in so far as I shall never be better," said one patient; "I feel as if there's no future," said another, yet at the same interview he said, "I dread everything; I picture all sorts of terrible things, that I shall go to asylums, and all sorts of things."

There are variations in the manner of description: there is sometimes conscious elaboration, but in the main, the uniformity in essentials is amazing. One variation is in the attitude towards recollections. Some insist on the remoteness and vagueness of the past, others on its sharpness and clarity in comparison with the present; they say they have lively pictures coming up:—

"Everything seemed suddenly to go backwards. The future seemed to go and the memory came forward. My memory became vivid, my childhood became vivid. It seemed to come automatically. I came to form mental pictures. From that I went back and back. It seemed as though everything was changing. The present and the future didn't worry me. Vivid pictures kept coming."

This variation is commoner in depressives, in whom the deterministic force of the past is strongly felt: things are occurring now, will occur in the future, which are only continuing evidences of past happenings; the future is closed to new action but an irrevocable process has been set in train. (In schizophrenics it is sometimes stated in the form, "I feel that what I say now will influence the future.")

This brings us to the depressives. Here emphasis must be laid on the inhibition or "retardation." This inhibition, which von Monakow calls "apathetic anastole," is a total change, affecting experience and determining conduct. I would bring into relation the altered time-consciousness which exists alongside unimpaired time-judgment (or time-perception, *sensu strictiori*), and altered activity-feelings which exist alongside unimpaired psychomotor capacity. "Retardation" is a personal experience, not an objectively demonstrable slowing or diminution of psychic or psychomotor performance; for this there is clinical and experimental evidence. In the case of the time-disorder, I have carried out on depressives a number of experiments in time-appreciation, using mainly the same tests as Bouchard set forth in his monograph, and I have found, as others have, ever since Revault d'Allonnes' work on the Korsakow syndrome in 1903, that the patients' professed incapacity to estimate duration is not borne out by objective tests for short intervals, and scarcely at all for longer intervals, though here other factors come in. I think that this inconsistency in both psychomotor and temporal experience can be referred to a common factor, which I should also hold responsible, in part, for that obstinate and ominous symptom—a belief that one has spent a sleepless night when it is not so: the distinction between this symptom and true sleeplessness is important and too often overlooked. In these depressive patients the rapidity with which time passes is felt, as well as its painful heaviness: the sufferer is beset with a press of memories, vivid and insistent.

The following is the account of his time disorder given by a recurrent depressive, self-reproachful and anergic, seen in his fourth attack:—

"I'm an absolute blank; I can't imagine what it is like outside. It seems as though the end is coming to me, dreadful. I can't imagine to-morrow. The days seem to fly by like

magic, but I can't look forward, I look backward all the time, all my childhood. I feel I can't do anything correctly, somehow."

Another, a young girl, said :—

"I can't think of the past and the future; it all seems a blank. Thoughts and memories will keep going round and round in my head: they are all of things I have seen and done during the last few years. They are like little pictures inside my head."

Only in so far as experiences acquire full meaning and reality, can they be incorporated into one's individual life history, and contribute to the development and unfolding of one's personality. It is inevitable that with the closing off of the individual future, i.e., anticipated activity, the past shall become torturing and intolerable: in a certain measure also the present. This is important in connection with compulsive states, to which I now come.

The intimate relation between compulsive and manic-depressive types of reaction is generally acknowledged, and is clinically and psychopathologically so manifest that it is unnecessary to consider the older view that one was a "psychosis" and the other a "neurosis," and that they were essentially different. The real problem is in many cases how to account for the development in, say, one depressive patient of delusions and in another of compulsions—compulsive thinking and compulsive acts.

Compulsive phenomena, considered not so much according to their content (which is symbolic), but according to their form, commonly show time components. This observation was made so long ago as 1874 in a paper in the *Journal of Mental Science*. As far as content is concerned, compulsive doubts and questions are sometimes taken up with time, especially in patients preoccupied with creation and causality, primeval chaos and suchlike metaphysical expense of spirit in a waste of thought. One patient, for example, with gross obsessional symptoms, said :—

"I keep worrying about the origin of God. I think about eternity. There is one thing that worries me: I can't get it off my mind. These eternal years trouble me a bit. My imagination is set going, and gets on numbers and eternal numbers, and eternity. Immensity, too. . . ."

Another patient with obsessional attacks of the cyclic variety said :—

"I have to reason everything out. I seem right up against a blank wall, and I feel I can't go on existing like that. . . . Do you think if I read some books about it would I get some rest? I have to just think and think about things like that. I can't get any satisfaction. I keep worrying about thousands of years ago, about the beginning of things. I feel I must know. If I don't know everything like that I don't want to go on. . . . I can't understand about the chaos that there was in the beginning and what was before that."

Time and eternity, the beginning of things and their end, are here the content of the compulsive phenomena; but what of their form? The main characteristic of this so far as compulsive thinking is concerned—and it applies equally to compulsive ideas and impulses—were referred to also in the foregoing examples: the inability to stop, to get satisfaction, the feeling of incompleteness, of lack of finality. One patient who said of her thinking "I feel as if I'm all mind, as if I'm mind altogether, and I don't get anywhere with my thinking," said also: "Sometimes when I am writing something I can't get past a full-stop. I keep making it bigger and bigger."

Many examples support the familiar view that it is in the incapacity to complete the particular act and so dispose of it, that the essence of morbid "compulsion" lies. Many psychologists now regard thinking also as an act, as internal conduct. Be this as it may, thinking and conduct are both carried out in constant reference to the future which is the arena in which the personality expresses or unfolds itself;

but this time-attitude is altered where general inhibition has occurred. For the normal person the past and the present are carried on into the future satisfactorily only when completed and dealt with; the less this is so the more there is of regret and return to the past: there is difficulty in completing an act, i.e., there is indecision, where the particular act will gravely affect the future. But in the inhibited obsessional patient it is not concern about effects in the future that hinders action; for him the future is barred, and so in his acts he is continually struggling against this check, hindered in completion and disposal, and thrust back to his starting point. Both the inhibited *depressed* patient and the inhibited *obsessional* do no effectual acts, but in the one it is by refraining from action at all, and in the other by indeterminateness and vain repetition of acts. I am sensible of certain difficulties in this and of its limited application; and I emphasize that one is here concerned with the form, not the content of the compulsions.

Compulsive phenomena of various kinds are prominent in encephalitics. In them, too, one finds disturbances in time-appreciation; thus a patient with oculogyric crises said :—

"My eyes turn up and I get sentences like 'What is what?' going through my mind. Time seems to go quickly. And yet it seems to drag. When I look at the clock the time has passed more quickly than I thought . . . . . It was one o'clock when I started reading and I'd read two pages by three o'clock and I expected it to be about half-past one . . . . . During these attacks I feel I can't see how I can come right. While I'm in it I seem to be thinking of the past the whole time, but these thoughts like 'What is what?' come in between . . . . . During the attacks I feel older: I feel as if I've had a lot of experience."

Another said :—

"Time seems to hang a bit but especially when it (the oculogyric attack) is coming on. I feel terrible, right down ill. You know it'll stop, but sometimes you feel it'll go on for ever. In the old attacks (more severe) used to feel you'd never get to the end of it."

I am omitting the toxic-organic disorders, in which there are much grosser disturbances of consciousness, especially evident in regard to orientation in space and time, where there is often not merely a change in time consciousness and time appreciation but also an ignorance of world time which rests on complicated disorders of judgment and memory. I am also omitting the drug-made time-disorders.

The large group of presenile or presbyo-phrenic patients presents some of the most striking of time-disorders. I do not regard them as a unitary or distinctive group but as showing combinations of the various reaction types. There are no features of this disorder ("involutional melancholia") beyond its greater frequency at this time of life—the climacteric and after—which may not be found in other age periods. There is nothing absolutely distinctive in the time-disorder; nothing which may not be found in younger people with similar nihilistic ideas, paranoid attitude and somatic hallucinations or delusions. I therefore quote without comment passages from a full account of striking time-disorder by a patient with "involutional melancholia":—

"Of course I realize I'm quite finished now: my faculties are all gone. I'm completely wrecked. I can't lose consciousness. I can't sleep, no sleep at all. I shall never really die . . . . . I know I shall be physically conscious for ever . . . . . I can't estimate time. I can't say what time it is because it's an artificial day; what you call a day with the artificial day is very much shorter than the ordinary day. The time goes very much quicker . . . . . I noticed my watch was accelerated . . . . . What I mean is this; since we had breakfast this morning, according to your time it is eight hours, isn't it? Well, we haven't had eight hours since this morning by Greenwich time. The time you keep here isn't Greenwich time. Yours is only a quarter of the real time . . . . . Probably in my months it'd be a couple of months since I came here, in what I call the ordinary time. But, of course, in your reckoning it'd be eight months, what you call a month . . . . . Time in the sense of being heavy on your hands is terrible here, I can't do anything. By Greenwich

time it goes very quick. But (considering) whether you find the moments interesting, time passes very slowly: every moment that passes is, you know, tedious and wearisome. Time in the sense of a period, though, is very quick. It would be about August, 1930, now by Greenwich time. I'm certain because I know Greenwich time couldn't have gone as quickly as July, 1931 (the date of the interview). The past seems a long way off, but that is only the tremendous tedium since. Figuratively speaking it seems years since I was out in the normal world . . . . . I never know any moment what is going to happen. It's the most terrible outlook I've ever had to look to. It's all perpetual. I've got to suffer perpetually."

There is little need for comment on that typical account. In some the change is interpreted, not in the personal sense, but, like the unreality, it is projected and the patient declares that she must die but that no one else will ever die.

There were schizophrenic elements in that patient's account of his experience. In some "involutional" patients they are obtrusive. What then are the disorders of time found in the schizophrenic? In addition to the usual unreality-accompaniments there are some distinctive features. One of these is the feeling of previous death and rebirth, as in this patient, a young woman:—

"I should say it was the beginning of a new world. I think I shall die at a certain time and live again. It seems as if I have died, not recently. I used to ponder over it, and think I'd been living a long time . . . . . I think there has been a break in my life . . . . . I feel things may slow up a little more; more evenly, that's all . . . . . I think the world stops and goes on so quickly that it's only noticed by certain people . . . . . In here the time goes much more evenly than anywhere else I know . . . . . Sometimes I've got up in the morning and wondered if it is night-time. It has seemed strange. I looked out of the window and saw the sun and it certainly looked like evening and it was really morning."

The feeling of an interruption in time occurs frequently in the records; thus one patient had said that "somebody was playing with the clocks." When talked to about it he said:—

"It's very bewildering. I don't seem to know how the date's going . . . . . It stands still for some time and then goes on again. I'm sure it does. Look at watch at half-past four and then look again an hour after and it's still half-past four. Guess a long interval has elapsed . . . . . Seems something wrong with the time here. It's later than it appears. Seem to sleep the night right round including the next day somehow . . . . . It seems as though a day's gone by and I'm still in bed . . . . . The elections were held on the 27th October" [actually two days before interview]. "I should imagine that was two or three weeks ago, judging by the days that have been missed."

Another, a young man, said:—

"There's a constant altering of perspective. I feel I'm not continuous. I seem to be reborn every moment. Although I remember things, I can't seem to exist to myself for a long time together. . . . I know that everything is the same as it always was. At odd moments I can reconcile my mind to the new. . . . I can see the continuity of events. . . . People's faces seem to open up to me fresh every time. I seem to have to arouse myself and accept the world again every moment. I have no continuity in my way of seeing things, and I have difficulty in bringing it back to a constant one."

This may be regarded as attributable to irregular changes in consciousness, i.e., occurrence of varying intermittent states of unreality; it has considerable bearing on other symptoms of schizophrenia. Another common feature in schizophrenia is the particular change in time experience often referred to as "thinking backwards," e.g.:—

"It's as though I had everything all mixed up with the present. Whenever anyone said anything to me, it referred back to some part of my life, numbers and colours. One mind was living back and one mind forward, living present-like. . . . I died three times; I

seemed to go right back. . . . I think it's the backward reflex of the brain. . . . I wish they would bring me forward in my mind. . . . I know I've got to die soon."

Another said :—

" I dream backwards. . . . I've run past myself."

Still others have anticipations of the " premonition " type :—

" When I am thinking about something, other people say just what I'm thinking. I think there must be some special significance."

Another :—

" Before a door opens I know it's going to open. I can tell what's going to happen."

Another :—

" Quick changes happen. I've noticed that when I do anything or when I'm going to do anything, people seem to imitate me."

Another schizophrenic patient, who has profound disorder of time-consciousness, though his personality is so far well preserved, and who weaves ingenious metaphysical speculations to account for these phenomena, declares that he is able to prophesy anything that may happen within the next few years ; this patient considers that the weeks this year are only as long as three days of former years : it is all controlled by a machine in the basement of the hospital which regulates the physical universe ; he has devised a new calendar to cope with the situation. In other patients, duration seems affected in the opposite sense : one said :—

" I can't believe there are twelve hours in a day. I live twenty-five years in one year."

The contradiction here is only apparent : the full statement would be, " I live twenty-five years of my time in one year of world time, your time." In others the fragmentation is impenetrable, e.g., " Seems as if something in the air went by, you see what's next going to happen ; puts me in distress. . . . What we've been talking, what we've been writing down, have to pull it up into Time, time, the clock." And once she wrote spontaneously the words " clock-time " though I had never used the expression to her. In these patients I could not demonstrate any objective time-disorder, again making use of Bouchard's tests.

A few words about mania and hysteria. In mania there is the subjective experience of capacity for rapid thinking and moving which is not confirmed by objective tests, with corresponding disproportion between personal time and world-time : " I can think faster, I do everything faster. I feel as strong as a lion. . . . I asked just now what time it was and he said quarter past eleven. I expected it was twelve . . . . . it seems more than twelve days since I came here." Another patient : " I think too quickly. Thousands and thousands of thoughts run through my mind. People don't seem to think quickly enough for me. I've always wanted to move quickly and other people move very slowly to my idea. I'm so full of beans and vitality. Time passes terribly quickly ; only thing is it seems the opposite ; at night I don't get to sleep and I long for life. . . . It seems rather long and monotonous."

In mania the not infrequent disturbance of consciousness manifests itself in misidentification of the environment and setting up of secondary temporal relations :—

" I know who you are ; you're Mr. Williams. You took my teeth out ten years ago."

In the speech and conduct of the patient there are frequent reminders of the playful fantasy activity of children, already referred to. With regard to hysteria ; in dissociation there may be a secondary time disorder ; for example, a patient with puerilism said :—

" People all seem grown up and older. And my Mum, I'm sure I know her from a long time ago. I look at her, but I can't make it out."

As to other hysterical manifestations, I may quote a patient who said when questioned about *déjà vu* :—

"Oh, yes, I've noticed that. Tell you what I do have. Horrible feeling of the bed moving. Lasts about five or ten minutes, seems ages. As soon as I close my eyes I feel I'm either drifting along or going round and round. I feel it'll go on and on, it's like a trance. It seems a long time while I'm in it."

Many hysterical patients report premonitions :—

"Many times I know what people are going to say just before they say it, and if I go up to a crowd in the street I know what's happened. Yesterday I was in the street and I said to myself, 'That lorry's going to turn over,' and that very minute it ran into a 'bus in Exwell Street and fell over."

These are, of course, not infrequent experiences in normal people, but when one asks hysterics routine vague questions about time, they commonly report this experience more frequently than other people. One of the striking features in the hysteric is that, although completely idle, many of them say that time goes quickly :—

"Time seems to pass very quickly, to me it do. I get up of a morning and it seems no time before I go to bed again,"

said a man with gross conversion symptoms, who lived a day as devoid of outward incident as sitting on a chair by the fire could make it. This is comprehensible if one considers that it is an inability to fill time with the contents of choice that makes for boredom, or a slow passage of time; in the hysteric these conditions do not obtain.

I have endeavoured to give a very brief if superficial account of the main features that have emerged in an investigation of rather wide range. The whole matter seems to me one of great importance in psychopathology; and though I would not be understood to refer all morbid phenomena to a disorder of time-consciousness, I think it appears in and colours most of the manifold changes of the functional unity, which we agree to subdivide into perception, affect, and the rest of our fictions. It is a primary alteration of consciousness and may be found almost as often as it is looked for in mental disorder.

Section of Therapeutics and Pharmacology.

[*January* 12, 1932.]

DISCUSSION ON THE THERAPEUTIC ADMINISTRATION OF OXYGEN AND CARBON DIOXIDE.

Professor J. S. Haldane: At the request of the President the subject of this discussion has been extended to cover the administration of carbon dioxide as well as oxygen, and I propose to open it by giving a short résumé from the physiological side of the effects of adding oxygen or carbon dioxide to the inspired air under different conditions, and then to refer briefly to some of the apparatus before us.

I need hardly remind you that in normal breathing of ordinary air the lung ventilation is so regulated that the partial pressure of carbon dioxide in the mixed alveolar air is maintained at a certain level, characteristic for each person, and usually corresponding to about 5½% of carbon dioxide in the alveolar air, reckoned as dry. If the supply of carbon dioxide brought by the blood, or the percentage of carbon dioxide in the inspired air is increased, the breathing is increased correspondingly, so as, if possible, to keep the carbon dioxide pressure in the alveolar air about normal. The increased breathing is mainly in the form of increased depth.

The oxygen pressure in the mixed alveolar is in this way kept at about 14% of an atmosphere, similarly reckoned. The latter pressure, if it were evenly distributed in the alveoli, would be sufficient to saturate the hæmoglobin of the arterialized blood to about 97%. Actually, however, it is not evenly distributed, even with the breathing normal, so that some alveoli have more and some less oxygen. The consequence of this is that owing to the shape of the dissociation curve of oxy-hæmoglobin the higher oxygen at some places does not compensate for the lower oxygen at others, and the mixed arterial blood has a lower oxygen-pressure than the mixed alveolar air, even when the breathing is normal. Owing to the very different dissociation-curve for the carbon dioxide in blood, there is no corresponding difference for the carbon dioxide in the alveolar air and mixed arterial blood. When the breathing is shallow and rapid, even though the volume breathed may be much increased, the deficiency in the mixed arterial oxygen-pressure becomes much exaggerated, and easily causes symptoms of want of oxygen. This was shown experimentally by Meakins, Priestley, and myself; also that the symptoms of shortage of oxygen, such as Cheyne-Stokes breathing and general discomfort, are relieved promptly by adding a little oxygen to the inspired air. There are various clinical conditions, such as bad cases of pneumonia or shock, in which the breathing becomes very shallow, or the distribution of air among the alveoli becomes irregular from mechanical reasons, and dangerous anoxæmia arises in this way, accompanied by cyanosis which is prevented when some oxygen is added continuously to the inspired air so that the available alveoli receive sufficient oxygen. Meakins and Davies have given a number of illustrative cases. It seems to me that the immediate cause of death in very many cases is want of oxygen due to shallow breathing owing to an enfeebled respiratory centre.

When breathing is under normal conditions, addition of oxgen to the inspired air has no noticeable effect. It is only when the blood is for any reason insufficiently saturated with oxygen that a marked effect is produced. The insufficient saturation may arise from other causes than irregular distribution of air in the alveoli, for instance, from a reduced pressure of oxygen in the inspired air, as in mountain sickness, or from a reduced percentage of oxygen, as in many industrial accidents, or, apparently, from exudation or thickening blocking the ready passage of oxygen through the alveolar walls, as in phosgene poisoning. In all cases, however, the condition of insufficient saturation, if at all marked, is a very dangerous one, with cumulative effects

on the respiratory centre itself, the higher nervous centres, the heart, and other organs. The one exception to this rule seems to be in chronic cases, where in some way not yet understood, the body has become acclimatized to the imperfectly oxygenated blood, and cyanosis is not accompanied by bad symptoms, nor does its disappearance on giving oxygen cause any relief.

When the blood becomes insufficiently saturated, the breathing is at first stimulated to a marked extent. But the increased breathing washes out too much carbon dioxide, and this soon neutralizes, to a large extent, the stimulating effect, so that it becomes scarcely noticeable even when consciousness is being lost. The breathing, however, becomes shallow and frequent, owing to enfeeblement of the respiratory centre.

The therapeutic administration of carbon dioxide is more recent than that of oxygen. It was first introduced generally by Yandell Henderson in the treatment of carbon monoxide poisoning in America, where cases are relatively frequent. Oxygen had hitherto been given in order to facilitate the expulsion of carbon monoxide from the blood, but it was found that when carbon dioxide was added to the oxygen the expulsion was greatly hastened owing to the increased breathing. Even if only air was used along with carbon dioxide the expulsion was faster than with pure oxygen. Another and I think very important factor comes in. I found that in an animal rendered helpless by breathing a certain percentage of carbon monoxide in pure air the symptoms were much ameliorated when expired air was substituted for the pure air without varying the percentage of carbon monoxide, or when about 5% of carbon dioxide was added to the pure air. The amelioration was evidently due to increased circulation through the brain, and I think that this effect, counteracting the depressant effect on local circulation of lack of carbon dioxide when the breathing is from any cause increased by want of oxygen, is a very important one.

Henderson has more recently laid great stress on another influence of carbon dioxide. When, after carbon monoxide poisoning, after serious operations, or under various other conditions, the breathing is feeble, it is apt to happen that a bronchial tube becomes blocked. The effect of this is that the air in the corresponding part of the lung is absorbed and collapse ensues which is apt to be followed by local pneumonia. This tendency is prevented by adding enough carbon dioxide to the inspired air to keep the depth of breathing normal. In new-born infants the expansion of the lungs is apt to be imperfect, followed by local pneumonia. The administration of carbon dioxide seems to be very useful in preventing this danger.

Another use for carbon dioxide is in getting anæsthetics quickly into or out of the body.

It seems probable to me that the therapeutic uses of carbon dioxide are likely to become even wider than those of oxygen, but only clinical experience can decide as to this. In any case it seems to me that both oxygen and carbon dioxide are powerful therapeutic agents in a variety of cases, and ought always to be as available as other therapeutic agents.

As regards apparatus of this kind for medical use, I should like to stress the importance of instructing nurses in its use. Want of such instruction leads to great practical difficulties and invites failures in what may be very serious emergencies.

Oxygen has often been given by primitive methods which are practically useless because the patient breathes hardly any of the oxygen. One of these methods is to allow a jet of oxygen to play near the patient's face, and another is to let oxygen blow through a funnel placed over the patient's face. Oxygen, or carbon dioxide, should be definitely administered in definite measured amount, which can be varied at once according to its effects on the patient, and which can be continued for days when this seems necessary. The aim is, not to merely delay death, but to tide the patient over a dangerous emergency until nature's recuperative powers get the upper hand.

Dr. Guy Crowden demonstrated the Drinker Respirator, which had been developed in America for the administration of artificial respiration for long periods of time in severe cases of asphyxia or paralysis of the muscles of respiration. The body of the patient is enclosed in an air-tight box, while the head protrudes through an adjustable rubber collar which fits closely round the neck, but does not hinder the circulation. By means of a pump driven by an electric motor, air is sucked out of the box and a negative pressure produced, which in turn produces a negative pressure inside the thorax of the patient, and inspiration takes place as in normal respiration. The next phase is the opening of a valve which admits air to the box, and the pressure returns to normal, whereupon the elastic recoil of the chest and lungs causes expiration. Positive pressure in expiration is not necessary because adequate ventilation of the lungs is produced by a negative pressure of 12 to 18 cm. of water, or 8 to 10 cm. in the case of babies, followed by a return to normal atmospheric pressure. The operator can control the depth of respiration by means of a valve.

Dr. Crowden said that the problem of resuscitation in cases of gas poisoning and other conditions of asphyxia had been systematically attacked in America. The prone pressure, or Schafer, method of artificial respiration had been universally adopted and oxygen and carbon dioxide used as a routine for cases of carbon monoxide poisoning. In New York, where suicides were frequent, the gas companies and electric companies had undertaken a resuscitation service, and every man out of some 35,000 employed by those concerns was thoroughly instructed in methods of artificial respiration. As a result of this action, the lives of more than 50% of cases of attempted suicide by gas poisoning were now being saved in New York.

In this country the number of cases of gas poisoning with suicidal intent had been rising for some time. Twelve years ago it was approximately 300. In 1929 the figure was 1,285 and, including accidental cases, the total number of deaths from gas poisoning in Great Britain was 1,530. The figure for London was approximately 240. This problem was surely sufficiently serious to warrant a more rapid application of the sound principles governing artificial respiration laid down by Professor Schafer and the universal use of carbon dioxide and oxygen, the value of which had been proved beyond question.

The apparatus now demonstrated is not designed for immediate first aid in cases of gas poisoning, but for very severe cases which do not respond satisfactorily to the normal methods of artificial respiration, or which require treatment for long periods. The machine has proved of particular value in cases of poliomyelitis in children, in which paralysis of the respiratory muscles developed. Treatment in the machine could be continued for days or weeks, until the muscles of respiration regained their power. A number of successful cases have been treated in America with the apparatus. I am convinced that it marks a tremendous advance in the saving of life in patients who otherwise would die in a few minutes. The absence of cyanosis in the patient is the indication to the physician that adequate artificial respiration is being given by the machine. Oxygen and carbon dioxide can be administered to the patient in the machine by means of a hood.

Dr. E. P. Poulton (President) said that the ordinary nitrous oxide gas mask, with valves and a large balloon, was found most effective. Recently he had had a case of severe bronchitis and heart failure, in which the administration of pure oxygen for fifteen minutes by this means quieted the patient, and no further oxygen was needed.

Dr. Reginald Hilton showed by means of graphs, the comparative effect of various methods of giving oxygen on the composition of the alveolar air. He also demonstrated a rubber cap to fit over the nose which could be used directly from an oxygen cylinder or from a Haldane-Davies water-valve if economy in oxygen was desired. For simplicity of use the nasal catheter was unrivalled; the nose-cap was

somewhat more efficient and was comfortable. The face-mask gave most oxygen to the lungs, but was not readily tolerated by patients suffering from pneumonia. The benefit of oxygen administration in pneumonia was not directly proportional to the amount given. Arterial blood-oxygen analyses showed that quite small quantities of oxygen, less than 1 litre per minute by a nasal catheter, removed much of the anoxæmia of pneumonia. More oxygen than this should be given however.

In some cases of pneumonia, carbon dioxide inhalation would increase the pain from pleurisy. Measurement of the tidal air, by the speaker, in a series of cases of pneumonia showed him that shallow breathing was not usually present. Indeed the tidal air was often increased. When we considered that part of the lung was solid, a normal tidal air meant over-ventilation of the rest of the lung. It would appear, therefore, that shallow breathing was not so important a cause of anoxæmia as had been thought. Shallow breathing would produce anoxæmia, but it had to be very shallow.

[Lantern slides were shown to demonstrate that in a normal person there was no reduction in the general alveolar oxygen pressure even when the breathing was diminished to half its normal value. Uneven ventilation could to some extent be compensated in anoxæmia, because better aeration in some areas tended to make up for poorer oxygenation in others, the arterial point being low on the hæmoglobin curve.

A case in which carbon dioxide administration had been found valuable was described. After a lumbar puncture in a patient with a positive Wassermann reaction, breathing almost stopped and the patient became intensely cyanosed, and unconscious. Carbon dioxide was given, at first continuously, and later at intervals. When it was stopped the breathing became extremely shallow and the cyanosis returned. Potassium iodide was given in large doses. After three weeks the breathing was again normal. It was thought that the patient probably had a cerebral gumma].

Dr. C. G. Douglas said that one difficulty which had to be faced by those who gave oxygen was lack of suitable apparatus. Obviously, the physician could not carry an oxygen chamber about with him, though one might carry around a Poulton oxygen tent. With Professor Haldane's apparatus he, the speaker, had during the War given oxygen continuously for three days to a case of pulmonary œdema resulting from phosgene poisoning. The mask was off for only five minutes an hour, and not at all during some hours. Luckily a good supply of oxygen was available in this instance. When treatment was commenced the man was already far gone. At first it was necessary to give 6 litres of oxygen per minute to abolish the cyanosis, but as the case improved the quantity of oxygen could be steadily reduced.

Some patients had fought against the mask, but with persuasion, and patience at the commencement of the administration of oxygen, he had secured its tolerance. In three minutes a nurse could be taught how to use the Haldane apparatus.

He agreed as to the value of administration of oxygen by the nasal catheter; he had seen it used by Adrian Stokes during a gas attack in France, and it seemed very valuable. He, the speaker, had found that with the Haldane apparatus, using the same flow of oxygen, the increase in the alveolar oxygen percentage was twice as great as when the nasal tube method was used. At first Professor Haldane made the mask of his apparatus small, so as to avoid the accumulation of expired carbon dioxide, as that might increase the ventilation of the lungs. Yandell Henderson then began to show the beneficial effects of the administration of carbon dioxide; so that the fears originally entertained about the undesirability of a "dead space" were now shown to be unwarranted.

The question of the administration of carbon dioxide opened up an interesting sphere. It was common to have such an apparatus in connection with mines in this country, where, of course, there was a risk of carbon monoxide poisoning. He was interested in the use of carbon dioxide in pneumonic conditions. First, in such cases there might be a failure of the respiratory centre; if so, one might succeed in

stimulating it by increasing the normal stimulus, and so tide the patient over his dangerous period. The work of Haldane, Priestley and Meakins showed that the distribution of air through the lung must be, under ordinary circumstances, somewhat irregular, some areas being under-ventilated, others over-ventilated. That would be emphasized when the breathing became shallow. Possibly there were ill-ventilated tracts of alveoli in the lung, and the failure of the blood passing through such areas could not be compensated by the over-ventilation of the other parts. One could not tell by measuring the tidal air what was the distribution of air in the different parts of the lung. Assuming that in some pulmonary complaints the reduced ventilation of the lungs might be an important factor and might lead to anoxæmia, there would then be an indication for increasing the depth of the breathing by such means as giving a carbon dioxide and oxygen or air mixture.

In a case of serious illness in which the breathing was depressed, though not exceptionally rapid or shallow, there was always the danger of stagnation in some parts of the lung, with perhaps, blockage of bronchial tubes by mucus and a collapse of those parts, with, maybe, some pulmonary œdema. He had seen a case which made him wonder whether such a risk could not be obviated by, at intervals, increasing the volume of the breathing by a natural process. If the movements of the lung were increased, air would be driven into the lung, the tendency to collapse would be abolished and the flow of lymph encouraged. In one case that he recalled, the right lower lobe was blocked up by a pulmonary embolus, and there were crepitations at the base of the other lung which aroused the anxiety of the physician. Oxygen was administered to the patient with benefit, and carbon dioxide and air was then tried. The administration of carbon dioxide and air was rapidly followed by a disappearance of the crepitations on the sounder side of the chest. Hence the question of trying to prohibit hypostatic pneumonia was an important one. He did not know how long a patient could be kept on carbon dioxide and air without difficulty. The hyperpnœa, if at all pronounced, became irksome even to a normal person after some time, and if one were dealing with a patient suffering from pleurisy the increased breathing might accentuate the pain. He would be inclined to give carbon dioxide and air at intervals for five minutes at a time, alternated with the giving of oxygen.

A striking case seen during the European War was one of very acute pulmonary œdema under circumstances when it was too dark to distinguish the colour of the patient's face. He took observations of the pulse, and there was nothing more striking than the change noted two minutes after oxygen was given. Administration could be safely carried on in darkness, the influence of the oxygen being judged from the condition of the pulse.

He would like to see this question of treatment by means of carbon dioxide and oxygen seriously considered by the profession. Physiologists knew the efficacy of these measures, but seldom or never had opportunities of trying them on human patients. The necessary apparatus could easily be transported to the patient's bedside.

Dr. M. S. Pembrey said he hoped that the therapeutic claims of oxygen and carbon dioxide would not be pressed too far; one must not be misled into thinking that pneumonia was chiefly a case of anoxæmia. He did not think the mortality from pneumonia would be reduced by the early use of oxygen or carbon dioxide, because, as far as he could see, all the indications showed that in the rapid respiration one had a sign of toxic, cardiac and febrile effects. One could not explain on the basis of anoxæmia the question of the definite period of an infectious disease. Years ago, Dr. Beddard and he (the speaker), by means of mask and valves, determined the ventilation in a case of capillary bronchitis with dyspnœa and cyanosis. Oxygen given in that case reduced the ventilation to a half. That was not found in cases of pneumonia.

The great value of oxygen and carbon dioxide was to tide a patient over emergencies. If he happened to be suffering from pneumonia he would regret the introduc-

tion of oxygen and carbon dioxide in the treatment from the beginning, as the slightest resistance from the valves caused discomfort in breathing. Nor did he think pneumonia or infectious disease would be cut short by giving these gases.

Dr. G. Marshall said that during the European War he had used the nasal catheter for the purpose a great deal, i.e., in gassed cases. Clinically the result was so good that he had been using the nasal catheter ever since, when giving oxygen therapeutically. With regard to pneumonias, a paper by Dr. J. J. Coghlan had recently appeared in the *Lancet* (1932, i, 13), pointing out the remarkable effect of inducing artificial pneumothorax in unilateral lobar pneumonia; the cyanosis diminished and the respiration-rate fell. He asked whether Professor Haldane would attribute that improvement to a reduction of the volume of blood circulating through the pneumonic area after the latter had been immobilized by the pneumothorax.

Professor Haldane, in reply, said he agreed that in the early stages of lobar pneumonia there were no distinct indications of want of oxygen, and there was no reason for giving it then, though he did not think it would cause harm. It was in the later stages that it would probably tide the patient over the crisis, and he would be likely to die if it was not administered. The effects recorded by various observers, particularly by Meakins, in these stages, were very striking, when the lips were becoming dull in colour and the patient was delirious and sleepless. With the giving of oxygen these symptoms disappeared Probably the oxygen did not shorten the duration of the pneumonia, but it enabled the patient to reach the crisis safely.

With most of what Dr. Hilton brought forward he was in agreement, and the results were very interesting, and to him, the speaker, new. But with regard to alveolar oxygen going up with shallow breathing, he did not think Dr. Hilton had taken in the main point of the paper by himself and his colleagues. Even if the depth was only cut down to half, it was very uncomfortable. Dr. Priestley went on with it longest, and it made him rather ill; but his alveolar oxygen was up whenever it was taken, and it was that which convinced him (Professor Haldane) and his colleagues that the distribution of air in the lungs was irregular, and that the alveolar air as taken in the ordinary way was only an average sample of what was in the lungs. Adding a little oxygen to the inspired air relieved the symptoms. At first they were surprised to find the alveolar oxygen up. When he, the speaker, lay down his breathing became deeper and slower, though this was not the case with all people. His went down from 14 to 7 or 8. If he set his breathing-rate by a watch at 20 per minute while in bed, he was amazed to find that the breathing became periodic, alternately becoming deeper and shallower. In Cheyne-Stokes breathing, produced artificially, there was always some anoxæmia. Cheyne-Stokes breathing occurred readily in himself, but was difficult to produce in Dr. Priestley. It was possible to have a raised alveolar oxygen along with distinct anoxæmia. That explained the apparent difference between Dr. Hilton's results and the clinical experience of others. He, the speaker, had been at loggerheads with the Copenhagen school for many years, owing to the assumption by the latter that the alveolar air was of even composition.

He agreed that the physician should give oxygen in the best way he could; with the nasal catheter or with the tube in the mouth, in fact by any convenient method of which he had experience. If, however, the apparatus described was used, one knew what quantity the patient was actually receiving, and that it was being given with maximum economy.

CORRIGENDUM.

Proceedings, xxv, 348 (Sect. Therap. 2).—**President's Address.**

Professor Greenwood's regression equation should read :—

$$\text{Wt.} = 0{\cdot}408 \times \text{Ht.} + 0{\cdot}693 \times \text{S.L.} - 70{\cdot}213.$$

(not "*plus* 70·213").

Section of the History of Medicine.

[*January* 6, 1932.]

The History of the Recognition of Tuberculosis as a Factor in Bone and Joint Surgery.

By E. Muirhead Little, F.R.C.S.

Consulting Surgeon to the Royal National Orthopædic Hospital.

The subject of this paper is, perhaps, not inappropriate in a year which is the jubilee of Koch's discovery, and due attention will later be given to the effects of this discovery. Long before the causal agent was known, however, pathologists and surgeons recognized the fact that scrofular strumous disease was of a like nature to pulmonary consumption, and earlier still there was a period in which this connection was unsuspected by the medical world. In recapitulating these stages, we shall see how first the microscope alone and then the new science of micro-pathology brought us to the state of knowledge which we enjoy to-day.

The ancient physicians such as Hippocrates and his commentator Galen knew and described tuberculosis in the lungs and also in the spinal column. They connected in some way the different affections, and attributed the disorders, pulmonary consumption and caries of vertebræ, to the effects of tubercles. "Tuberculum" means a growth or lump or knob, and is the Latin equivalent of *φῦμα*, the term used by Hippocrates and Galen. The latter wrote as follows (third Commentary on the treatise on the joints):

"But some think that the spine is twisted (or diverted) backwards when great, hard and persistent tubercles [magna, dura ac diuturna] have begun to grow in it, by the burden of which (tubercles) the vertebrae are driven backwards." . . . and again: "But that the spine is driven backwards by growing tubercles, Hippocrates himself shows a little further on."

We may ask what was the nature of these tubercles, which caused such results and were recognized as akin to formations found in the lungs. Is it not possible that the tubercles so plainly described as large, hard and persistent, were no more than the cutaneous thickenings, sometimes covering a bursa, which form under the influence of pressure and friction over the ends of the spinous processes, etc., in a case of angular deformity, and which, when the thoracic vertebræ are the seat of disease, are not far removed in actual distance from the contents of the thorax? If this suggestion seems far-fetched, I venture to think that it is less fantastic than that which would have us suppose that, without microscopes or chemical reagents and with not too accurate notions of human anatomy, these observers distinguished tuberculous granulomata, and yet did not describe their appearance. In a good many mentions of tubercles in the literature from Galen to Nélaton, there are no satisfactory descriptions of their appearance or structure. The fathers of medicine gave us no description of tubercles in other parts of the body, and it would almost seem as if they only recognized their presence in the thorax.

From the second century of our era, when Galen wrote, to the sixteenth, when Jacques Dalechamps of Lyons annotated his works, is a far cry.

Dalechamps, as an editor of Galen and a re-discoverer of caries of the vertebræ, deserves some extended notice. Born at Bayeux in Normandy, 1513, he took his M.D. degree at Montpellier in 1547, and settled at Lyons five years later. There, according to the *Dictionnaire des Sciences Médicales*, he devoted thirty-six years to

the practice of medicine, in which he distinguished himself by brilliant success and acquired a great reputation. He united to a profound knowledge of the dead languages great erudition, as was shown by his translations and in his commentaries on several Greek and Latin authors. He published, in 1552, *De Peste, Libri Tres*, in 1586 *Historia Generalis Plantarum* and, in 1570, *Traité de Chirurgie*, of which three editions appeared, the third of which was published in Paris in 1610 under the title of *Chirurgie Françoise*. A copy of this edition is in the library of this Society, and contains Dalechamps' translations from Galen and annotations thereon which originally appeared in 1566. In one of these annotations he refers to displacement of the vertebræ by violence, and goes on to say,

"the other kinds arise from external and internal causes. . . . The internal causes are only to be stated as by Galen in his commentaries on the third book of the Dislocations and on aphorism 46 line 6, as cold swellings developed on the ligaments of the vertebræ."

Further, he says:—

"Sometimes also these swellings finish with a long dysenteric flux, when they suppurate and the suppurated matter passes into the bowels and makes them sore [literally, 'scrapes them', 'les racle']. These patients are subject to disease of the kidneys and bladder and to abscesses which are chronic and difficult to cure, which point in the loins and groins by sympathy and communication of these parts with the diseased ones . . . as often happens in parts which are of the same nature and substance; or which are adjacent. . . . The legs become more slender, the beard and pubic hair appear late and are scanty. They (the patients) are less than normally fecund. . . . When the vertebræ of the neck are displaced angularly and principally the second and first, all the parts situated below lose sensation and movement; but if they are displaced in a rounded form, movement and sensation in the parts below are little or not at all affected. If they (the vertebræ) are displaced obliquely. paralysis appears on the side of the displacement in the part which receives the faculty of movement and sensation by the branch of the compressed nerve and on the opposite side there are spasms. And these accidents happen more in distortion of the cervical vertebræ; less often if the twist is of the vertebræ of the chest."

Dalechamps goes on to give a good clinical picture of a case of severe caries of the vertebræ with paralysis, thus anticipating Pott.

Richard Wiseman appears to have been the first to use the term "White Swelling" for chronic tuberculous disease of joints, and especially of the knee-joint. In his book, *Severall Chirurgicall Treatises*, dedicated to King Charles the Second, to whom he was Sergeant-Surgeon, and published in 1676, Treatise IV is devoted to the King's Evil. As Sergeant-Surgeon, one of Wiseman's duties was to examine applicants for cure by his sacred Majesty's touch and certify that they suffered from scrofula. In Chapter I, speaking of the efficacy of the Royal touch, he says:—

"I myself have been a frequent Eyewitnesse of many hundreds of cures performed by His Majesty's touch alone"

and he speaks of the

"great concourse of strumous persons to Whitehall and the success that they find in it."

Struma generally at that time seems to have meant enlarged glands more often than any other malady, but among cases touched were sebaceous cysts and various other non-tuberculous affections. Spina ventosa is included among strumous diseases. White swellings are described as "Swelling affecting the joints in this disease, (i.e.) the King's Evill." He describes one case which seems to have been undoubtedly tuberculous, in which, in a child, there was

"a Strumous Ulcer of the outside of the ankle and Caries in the ends of the fibula and three of the Spondylls of his back distorted."

Auguste Nélaton, in his *Recherches sur l'Affection Tuberculeuse des Os*, published at Paris in 1837, refers among many other authorities to a work by Marcus Aurelius Severinus, *De Recondita Abscessuum Naturâ*, published in Latin

at Naples in 1632, in which he speaks of tubercles as the cause of spinal disease and deformity. After quoting Galen, as all other orthodox physicians at that time must do, he affirms on his own account the cause of hunchback to be tubercles, in the following terms:—

"But when you come to look at the causes, some hunchbacks are accidental, some due to a *tuberculum*; now of tubercula some are colder, some mild, some hotter [literally 'more cooked'], some hard, some not fully formed, and they either come in the neck or the thorax or in the loins or even lower. I at any rate insist that a hump (or gibbosity) is due to a tuberculum if you accept what Galen says about it when in Note III 'de Articulis' he stated that the origin of it must be due to some tubercula in the spine, and not in the lungs as Mercurialis wrongly thinks."

Severin, like Dalechamps, anticipated Pott in his account of disease of the vertebræ, complicated by paralysis.

Centuries of clinical observation had established in the minds of Wiseman and his contemporaries a firm belief in the symptom-complex known as scrofula or struma and though their diagnosis was sometimes at fault and, like Wiseman, they sometimes included under "King's Evil" diseased conditions which were in no way tuberculous, they had evidently a distinct idea of what was meant by the strumous or scrofulous diathesis and associated pulmonary consumption, chronic joint disease, enlarged glands and caries of the vertebræ with one another.

In the second volume of the works of Joh. Zacharias Platner will be found a Prolusion, No. XXII, by Ernest Frederick Haacke, dated 1743, and entitled: *De iis qui ex Tuberculis Gibberosi fiunt*; it is illustrated by two plates, which show caries of the lower dorsal vertebræ in a child, and are apparently drawn from a macerated specimen. The author also describes a case of caries of the dorsal vertebræ with almost complete destruction of the bodies of four vertebræ.

This essay is quoted here merely to call attention to the author's confident assumption that caries of vertebræ is caused by "tubercula," whatever that may be.

Percival Pott's celebrated *Remarks on that kind of palsy of the lower limbs which is frequently found to accompany a curvature of the spine* were published in 1779. Pott has very little indeed to say about pathology, and only once makes any reference to scrofula in this essay. It seems to me quite doubtful if Pott had any clear idea of the sequence of events leading to caries, deformity and palsy, for he says:—

"previous both to the paralytic state of the legs and to the alteration of the figure of the backbone, there is a predisposing cause of both, consisting in a distempered state of the ligaments and bones, where the curve soon after makes its appearance."

Pott's "remedy for this most dreadful disease consists merely in procuring a large discharge of matter by suppuration from underneath the membrana adiposa on each side of the curvature, and in maintaining such discharge until the patient shall have perfectly recovered the use of his legs."

Pott condemned the use of "steel stays, the swing, the screw chair and other pieces of machinery."

A surgical advance was made by a surgeon in Liverpool, that city which was later to be the scene of so much activity in the surgery of bones and joints. In 1783 there appeared *An account of a new method of treating diseases of the Knee and Elbow* in a letter to Mr. Percival Pott by H. Park, of Liverpool, one of the surgeons to the Hospital, dated London, 1783. The writer described a new method of treating "Scrophulous affections of the joints, commonly distinguished by the name of 'White Swellings,'" by excision of the bone ends.

Park recorded two very successful cases. His first operation was performed in 1781. Similar excisions had been devised by P. Moreau, M.D., of Bar-sur-Ornain, but had not been published until after Park's letter to Pott. His observations and cases were, however, published at Glasgow in 1806, with observations by James

Jeffray, M.D., Professor of Anatomy and Surgery in the College of Glasgow, and were illustrated by engravings.

In a prize essay published in Berlin in 1797, and entitled *A treatise on the Nature, diagnosis and cure of Scrofula*, C. G. Hufeland discussed disease of the vertebræ, scrofula of the articulations and of the joints and bones, and white swelling. He mentioned tubercles as present in some of these conditions.

Bryan Crowther, who was Surgeon to Bridewell and Bethlehem Hospitals, writing in 1797, emphasized the value of rest in the treatment of joint disease. In a pamphlet of 122 pages, *Practical Observations on diseases of joints, commonly called white-swelling*, Crowther showed that he was before his time in some of his views, although he does not refer to tubercle. He mentions the essential unity of white swelling, spina ventosa and phthisis pulmonalis.

In 1794, in his *Observations on the disease of the Hip-joint* (London) E. Ford reaffirmed the connection between hip disease, white swelling of the knee, caries of the wrist, etc.

In 1802 John Herdman published at Edinburgh a dissertation entitled *An examination of the grounds on which white swelling of the joints has been divided into a scrophulous and rheumatic species.*

This must be one of the first attempts to establish a differential diagnosis of diseases of joints. He argued that scrophula or white swelling does not depend on plethora and a phlogistic diathesis. He condemned bleeding, cold baths and sea-bathing, saying that at King's Lynn, where the inhabitants bathed much in the sea, scrophula was rife. He recommended warm baths, Bath waters and camomile flowers. As to local treatment he held what we now hold to be sound opinions, namely, that

"there is perhaps nothing so injurious as motion of the joint," and he enjoined absolute rest and support by a flannel roller. In abscess "the free incision produces the very worst effects." "Scrophulous abscesses . . . should never be opened, but when they press on some important part, or when life is in danger by their bursting internally; but in common cases, when they burst spontaneously, and when the constitution is properly supported they generally heal, though it is a tedious process." "Hectic and every untoward symptom supervene on the opening of large scrophulous abscesses."

In summing up, he says: —

"In the treatment of white swelling, therefore, surgery is almost altogether, if not altogether, out of the question."

Evidently Herdman was a conservative surgeon, who would have objected to the implication in the title of this discussion.

With Benjamin Brodie's *Pathological and Surgical Observations on Diseases of the Joints*, published in 1818, we come to another attempt to distinguish and record the morbid anatomy of joint diseases. He does not lay any stress on the connection between white swelling and scrofula and tubercular disease in the lungs, nor does he apply the term "tubercle" or "tubercular" to the deposits in the joints, which he described. No doubt we should now class some of his cases as osteomyelitis. His treatment was in general conservative.

Two years after Brodie's book appeared, James Wilson, F.R.S., delivered a course of lectures before the Royal College of Surgeons in London on the Diseases of the Bones and Joints, these were published in 1820. Wilson was Professor of Anatomy and Surgery at the College and Lecturer on the same subjects at the Hunterian School in Great Windmill Street. He gave a very clear account of chronic joint tuberculosis and, referring to scrofula generally and white swellings of joints, he says:—

"The tubercles formed in the lungs have strong characteristic marks of being allied to this disease; it affects many of the abdominal viscera primarily and collaterally; so does it the brain and the membranes which contain that organ."

In his views as to the value of rest and fixation and as to the choice of positions for ankylosis and in treatment of abscesses Wilson showed himself an enlightened surgeon. As for caries of vertebræ, he thinks that instruments may be useful by extending the spine so as to assist in supporting the weight and "will prevent diseased surfaces pressing so much on each other."

Sir William Watson Cheyne, in his book on *Tuberculous Diseases of Bones and Joints* very truly says that "the real starting point of the study of tubercle in bone is Nichet's work, published in 1835." This appeared in a series of articles in the *Gazette Médicale de Paris* and was entitled *Mémoire sur la nature et le traitement du Mal Vertébral de Pott, par M. Nichet, chirurgien en chef désigné de l'hôpital de la Charité à Lyon.* He says that Delpech had long professed the opinion that Pott's disease was due to a tuberculous affection of the vertebræ, and this Nichet considered to be proved true by a great number of observations. Nichet says:—

"Des dissections fréquentes m'ont permis de voir les tubercles des vertèbres sous toutes les formes. Je les ai suivi dans toutes les points de la colonne vertébrale, dans les divers degrés de leur développement; j'ai pu étudier l'influence qu'ils exercent sur le rachis et sur la moelle; il m'a été donné de saisir les rapports non encore aperçus entre les symptomes et les altérations anatomiques."

He concludes that the changes in the spinal column which accompany Pott's disease have as their origin the scrofulous tubercle. As for phthisis, he holds that "it is through the lung that most of those attacked by Pott's disease die,"

a conclusion not generally accepted since.

"Moreover, tubercles may be met with in all parts of the economy; our observations have shown them to us in the peritoneum, omentum, mesentery, prostate, the intermuscular cellular planes, the loins, the abdominal walls, the thigh etc."

Although Nichet was so up to date in his recognition of tubercle he was rather old-fashioned in his recommendation of bleeding, cauteries and issues. His only other recommendation was rest in the horizontal position.

The tubercles of Nichet seem to be very different from the "great, hard, persistent" tubercles of Galen.

In his *Recherches sur l'affection Tuberculeuse des Os*, Auguste Nélaton has furnished us with a store of information on the history of joint and bone disease. He says with truth of tubercle of bone that

"known in almost all times by some doctors, and at the same time ignored by the greater number, it is one of those diseases for the description of which one will seek in vain in the dogmatic treatises on surgery."

But Nélaton quotes from Platner a dissertation of 1735 which, as he says,

"shows that the existence of vertebral tubercles was generally known and the production of gibbosities was attributed to them. In speaking of a child who was treated by Wedelius and recovered with an angular deformity of the spine, the author of the dissertation wrote:

'Vero enim simile videtur hunc puerum ex tuberculis intus enatis gibberosum factum fuisse, tandemque paralyticum.'"

Nélaton summed up his historical account as follows:—

"The knowledge of this disease goes back to a very remote epoch, although authors have rather pointed it out than described it. That the gibbosity (the hump) has always been the point of departure of their researches, and that almost all that they have said is applicable exclusively to tubercles of the vertebræ."

In 1844 Rokitansky introduced modern pathological ideas when he definitely stated that there was similarity between chronic disease of joints and "tubercular" disease of other organs, though the honour of having first demonstrated the existence of a tuberculous nodule in joint disease is claimed for Köster in 1869.

Watson Cheyne, in his book (2nd edition, 1911), says :—

"Köster, in 1869, was the first to study these diseases of joints histologically, and to recognize fully their tuberculous nature."

In 1862, when the first edition of Holmes's *System of Surgery* appeared, the influence of the German advances in diagnostic pathology had not made itself felt in this country to any extent. The editor, Mr. Timothy Holmes, wrote on diseases of the bones as

"Specific diseases which occur either in modifications of the inflammatory process, syphilis, scrofula, rheumatism in bone, or as constitutional conditions leading to changes in the bony structure."

In this *System* the article on Orthopædic Surgery was written by Dr. W. J. Little, Physician to the London Hospital. It included in its scope the deformities resulting from joint and bone disease, but not the treatment of active disease: tuberculosis as such was not included. When it was introduced in the guise of Pott's disease or joint contraction it was treated as such.

Before the third edition of the *System* appeared in 1883 great changes and advances of knowledge had occurred. Antiseptic surgery had made operations less hazardous; Koch had found the tubercle bacillus and had convinced many surgeons of its importance. But the editor stated in a footnote: "Tubercle in bone is, in any view of the case, rare."

The writers on bone and joint disease and scrofula in this edition, such as the Editor, A. E. Barker and Frederick Treves, were very cautious in dealing with Koch's discovery. The last-named expressed very conservative opinions as to scrofula. While allowing that white swelling was a form of tubercular disease of low virulence he argued

"that facts show, on the one hand, that although scrofulous patients do exhibit tubercle, the bulk do not die of tubercular disease and, on the other hand, that local tubercular processes do not of necessity call for immediate extirpation, inasmuch as they are capable of undergoing a safe and spontaneous cure."

Some other surgeons were less prudent than Treves and, under the supposed protection of Listerism, Park's operation of excision had been revived and rather freely practised as well as the later procedure of arthrectomy. In 1886 there appeared *Diseases of the Joints*, by Howard Marsh, F.R.C.S. He fully accepted the discovery of the bacillus but maintained that taken early and treated conservatively, a cure was general y assured in the joints of the hip, knee and ankle.

Orthopædic surgeons, not only in England, had some time earlier arrived at similar conclusions and, believing that surgical tuberculosis was usually a chronic affection, they had not, except in rare cases, subjected their patients to the radical surgical measures of which Sir W. Watson Cheyne wrote, and had avoided the phase of early excision of joints, arthrectomies and such-like measures.

At the National Orthopædic Hospital, when I was first connected with the staff in 1880, those suffering from chronic joint diseases, including Pott's disease, were treated as either out- or in-patients. They were treated as far as possible by rest, fixation and extension. The mild nature of the disease under such treatment is shown by the fact that I was able to report in 1892 that, as regards Pott's disease, out of 133 in-patients there were 10 cases of paralysis and 8 of paresis, all of which recovered. As regards abscess, there were 21 cases in 133 in-patients and 7 in 187 out-patients.

By the use of such appliances as Bradford's frame, it was possible to treat cases satisfactorily as out-patients, and from 1892 onwards all my tuberculous in-patients lived night and day on the balconies in the open air, even during London fogs. I have no doubt that equally good results were obtained at other orthopædic hospitals

and at special hospitals where treatment was conservative, as at the Alexandra Hospital, under Mr. Howard Marsh.

The open-air movement took a great step forward when, in 1900, Miss (now Dame) Agnes Hunt started the open-air hospital at Baschurch in Shropshire, and when Mr. (now Sir) Robert Jones interested himself in her effort and became one of the warmest advocates of sun and air. Other open-air hospitals followed. As the quotation from Sir W. Watson Cheyne will show, the enthusiasm for radical operations in joint disease had waned by 1911. At present the expectant or conservative treatment is generally accepted and advocated in all the hospitals for cripples throughout the country, but some surgeons advocate and practise ankylosing operations designed to shorten treatment.

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[*February* 3, 1932.]

Alfred Higginson and His Syringe: with a Side-glance at the Clyster.

By DAN MCKENZIE, M.D.

ON the Continent of Europe the rubber enema-syringe, that indispensable article of modern medicine, has for long been known as "the English syringe"; and correctly so, since it was Alfred Higginson, of Liverpool, who in the fifties of last century devised its compressible reservoir and its ingenious system of valves, comparable with, and possibly suggested by, those of the heart.

The anal route to the bowel had, however, been used in the treatment of (usually local) disease for thousands of years, and, as we shall see in a moment, it has been employed also by a number of nature-races.

The words "clyster" and "enema," by the way, seem to be used synonymously. Etymologically, however, their meaning is by no means identical, since "clyster," from *κλύζω* hence *κλυστήρ*, means a "washing-out," or "douching," as we would say; whereas "enema" (*ἔνεμα*)—a word that goes back as far as Dioscorides in the first century A.D.—means merely an "injection," as it is derived (they say) from *ἐνίημι*, "to throw in." Thus the two words mean quite different things and might be used as distinctive terms even to-day.

According to Herodotus (Book II, 77), the ancient Egyptians used to purge themselves for three successive days every month by means of emetics and clysters

(κλύσμασι); and he uses the word κλυστὴρ in describing how cedar oil was injected through the anus into the bowel in one of the methods of embalming, the word being usually translated into English by "syringe," although perhaps "clyster-pipe" would be more accurate (Book II, 87). In the Ebers papyrus also, there are some half a dozen formulæ which seem to have been used as enemata, as the directions instruct the physician to "inject" or to "pour" them into the anus.

Thus the practice is ancient. It is also, in accordance with the general rule, very widespread, even if it be not, so far as we know hitherto, very prevalent over the surface of the globe. Thus the Mayas of ancient Central America and Mexico are credited with a knowledge of enemata, and Bartels reports that the more recent Dacota Indians were known to employ them, using syringes made expressly for the purpose, and injecting decoctions of various kinds. An enema tube is in use among the present-day Indians of South America.

In modern Liberia, the negroes use a calabash shaped like the neck of a bottle, apparently "pouring in" their remedies as the ancient Egyptians may have done, and the same instrument is used in other West African countries. In Persia, there is a regular domestic vessel for the purpose, described as a long funnel with a rounded nozzle bent like a catheter. It is made of glass or, in the richer houses, of silver, and can be taken apart for cleaning.

The clyster was one of the regular methods of Greek therapeutics, and, according to Francis Adams, both Greeks and Romans used a syringe, that is to say, an instrument with a cylindrical barrel and a piston, but apparently not furnished with valves.

In addition to those two varieties of instrument for the administration of clysters, we also find collapsible bags, consisting usually of the bladder of an animal such as the ox, and provided with a nozzle. This, or a bag of silk, was the ordinary apparatus of Renaissance medicine, as figured in the old books (e.g. Fabricius Hildanus), the bladder being sometimes furnished with both inlet and outlet tubes, each with a stop-cock.

It would be natural to suppose that the collapsible bag was probably used for the administration of liquid in bulk, while the rigid metal syringe was reserved for local remedies, since these are, generally speaking, small in quantity. But there does not really seem to have been any rule in the matter, for, on the one hand, Fabricius Hildanus used the bag in the introduction of his local remedies for dysentery; and, on the other, the specimens from the Wellcome Museum now on view include metal syringes large enough to hold two pints of liquid and even more.

As may also be seen from these specimens, much ingenuity has been expended in the past in disguising the syringe so as to avoid offending delicate susceptibilities, and it will be readily acknowledged that not only are they more elegant in appearance, but they must also have been more easy for an unaided patient to manipulate than our modern pattern is. It is, therefore, rather surprising that they have so completely disappeared. Most of the varieties apparently emanate from the Continent and from the century immediately preceding Higginson's time. Some of them, neatly camouflaged in mahogany cases, were evidently constructed to be travelling companions.

In France about this time (the end of the seventeenth and the beginning of the eighteenth century A.D.) recourse to enemata assumed the characteristics of a medical, and even of a society, craze. The fashion, like that of wearing wigs, was due, it is said, to Louis XIV, who, like the ancient Egyptians, made a frequent use of the clyster for hygienic reasons. The fun made of the practice by Molière is known to all the world.

But it is more interesting to us as medical historians to turn to the contemporary writings of the Dutchman, Regner de Graaf, whose *De Clysteribus*, published in 1668, gives a reasonable and temperate account of the therapeutic use of the

clyster. Among other points of practical importance he has the acumen to raise the question as to whether nutrient enemata can be absorbed from the rectum or colon, and answers it in the negative. On the other hand, however, he considers that stimulants so administered do exercise a definite effect. These, and indeed most of his opinions, would probably be accepted by physicians at the present day. De Graaf, as a matter of fact, is amazingly modern in his outlook.

The rubber syringe, like a thousand other articles of the same material, is a product of our own civilization. But, curiously enough, the chemists who first taught us how to convert the raw natural rubber into a tough yet flexible material have apparently been forestalled by savage races in the rubber districts of South America.

Our authority for this is Baron E. Nordenskiöld (*Journ. Roy. Anthrop. Institute*, vol. lix, 1929), who reproduces from a photograph the rubber enema syringe used by the Amerindians of Guiana and the Amazon. We naturally wonder whether the manufacture of this instrument may not have been suggested to the natives by some European, but Nordenskiöld says: "It is to Indians that credit is due for the discovery of rubber, and its utilization in the form of rubber balls, enema syringes, waterproof fabric,[1] elastic rings, etc.," and "In pre-Columban times the Indians were acquainted with all the qualities that make india-rubber so valuable in modern industry."

The Amerindian enema syringe consists of a simple rubber-bag, somewhat oval in shape, with a conical nozzle, well enough adapted for the use to which it is put. There seem to be no valves, and it is filled and emptied by its single orifice, like the "squirt" of our boyhood.

Increasing acquaintance with the devices of nature-races generally has shown us that their inventive powers are less meagre than was at one time supposed. But the discovery of a rubber enema-syringe in the depths of Brazil is surely as astonishing as anything hitherto reported, testifying, as it does, not only to considerable medical knowledge but also to technical skill of a high order.

Before Higginson then, we may say there were three varieties of instrument; first, the rigid receptacle, of pottery, metal, or glass, with, originally, a rigid nozzle, which has developed into our douche-can; second, the simple metal syringe, apparently unvalved, and so we cannot call it a pump; and third, the collapsible bag, whether it be an animal's bladder, or of silk, or the unique rubber squirt of South America. The first has a double opening with an anal nozzle, the liquid being poured or run in under atmospheric pressure simply, or perhaps being blown in; and the second and third are actuated by manual pressure. Thus, Higginson's syringe combines the advantages of all three as the liquid can be forced in under pressure and in any quantity. There is no doubt that it was a quite original invention.

A brief sketch of Higginson's career, position, and character will not be out of place here, particularly since he seems to have hitherto escaped the eagle eye of the medical biographer.

Alfred Higginson was a well-known surgeon on the staff of the Liverpool Southern Hospital (now the Royal Southern Hospital) from 1857 until 1867, and Consulting Surgeon till his death in 1884 (see later).

From contemporary medical directories we learn that he qualified M.R.C.S. and L.S.A., and L.M. (Dublin) in 1832. He applied for registration as a medical practitioner in 1847, and his form of application may still be seen at the offices of the General Medical Council. For a time he was demonstrator of anatomy at the Liverpool Medical School, and he contributed an article on the knee-joint to the *Cyclopædia of Anatomy and Physiology*. Unfortunately, I have not succeeded in

1 The Edinburgh surgeon, James Syme, father-in-law of Lord Lister, is said to have been the first to dissolve rubber and to paint it on fabric to make a waterproof material; but the process was patented, not by him, but by Mackintosh.

discovering when he invented his famous syringe, nor have I been able to find its first published description, if, indeed, any such description ever was published. All I have found, as may be seen from the accompanying figure from an old surgical instrument catalogue, is that its reservoir was originally cylindrical, or, as it was described, doubtless by Higginson himself, in his notice in the *Medical Directory*, an "elastic barrel."

A further point of interest in Higginson's medical life is that in 1857 he reported, in the *Liverpool Medico-Chirurgical Journal*, seven cases of blood transfusion, the blood being propelled through a syringe valved like his enema syringe (lucky the blood did not coagulate !)—an early example of vein-to-vein transfusion.

Further instances of his ingenuity are manifested in an ether inhaler, a stomach pump, and an enema syringe without valves or stop-cocks. (What has happened to this last ? It sounds attractive.)

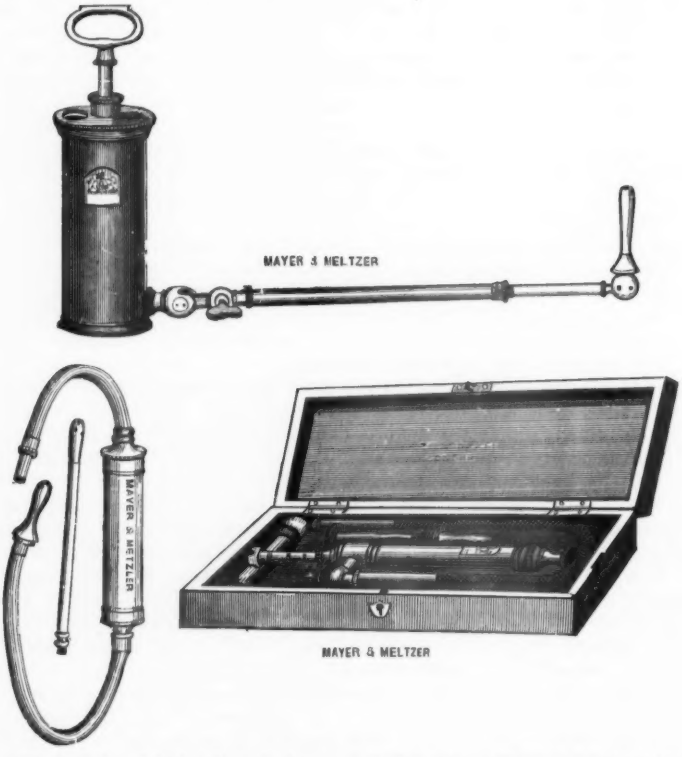

FIG. 1.—Higginson's original pattern is shown on the left below. The upper syringe (shown below in its box) is the French pattern that preceded Higginson's. (Courtesy of Mr. Mayer.)

I may here mention the coincidence that Regner de Graaf, of whom we have already spoken, also practised transfusion, but only from animal to animal, using a tube composed of a bird's intestine, to each extremity of which was attached the quill of a duck's, or some other bird's feather. The quill, he says, could be introduced into the blood-vessels without difficulty.

It is interesting to note how the hollow viscera of animals were used by them of old where we use rubber.

The following extracts from letters give some additional details of Higginson's life, the most interesting of which is his connection by marriage with Harriet Martineau. The first, dated 1924, from his nephew Charles G. Higginson, was sent to Dr. C. J. Macalister, of Liverpool, to whom I am indebted for permission to publish it. The second, with its vivid description of Higginson's personality, was written (in 1930) by Dr. Henry Harvey, to whom I extend my grateful thanks.

"Alfred Higginson, the second son of the Reverend Edward and Sarah Higginson, of Heaton Norris, near Stockport, was born in Heaton Norris in May, 1808, and died at Tulse Hill, London, in 1884, having retired there in 1878. He was buried in Lower Norwood Cemetery, London." (No notice of his death seems to have been taken in any of the contemporary medical journals.—D.M.)

"He was long on the staffs of the Liverpool Southern Hospital and the Liverpool Children's Hospital. He was an adroit surgeon, and gained a high local reputation for his skill in the transfusion of blood.

"He married Ellen, youngest daughter of Robert Martineau, and sister of Harriet and the Reverend James Martineau. Three children died in infancy, but one daughter, Harriet Emily, lived till 1917, when she died at Harriet Martineau's house, The Knoll, Ambleside, aged 72, and one son, the Reverend Philip Martineau Higginson, still survives aged 77.

FIG. 2.

ALFRED HIGGINSON, M.R.C.S., L.S.A., L.M.
Of 44, Upper Parliament Street, Liverpool.
1808-84.
Inventor of the Higginson Syringe.

"The enclosed photograph (fig. 2) was obviously taken in old age. I have also a painting of him, at perhaps the age of 40, with professional 'mutton-chop' whiskers, but otherwise shaven. Both this photograph and Mr. Higginson himself were constantly mistaken for Charles Darwin; all the same, one would think the faces were different enough.

"Please tell me if you would like to see the painting mentioned above. But the family never liked it, and certainly the photograph shows him looking like the grave and noble and competent old gentleman that he really was."

The following is from Dr. Henry Harvey :—

"Dear old Higginson! Yes, I knew him well, if not intimately. When I came to be Senior House Surgeon at the Royal Southern Hospital I was much struck by his personality. A man decidedly below the average height, massive overhanging eyebrows, with keen eyesight and still keener insight; nothing escaped him. So like the portraits and busts one has seen of Darwin that I fancy one might easily have personated the other. This resemblance had, I understood, been brought to Darwin's notice, and he had recognized the truth of the marvellous resemblance. The type of mind was very similar. I think the word 'meticulous' in all things best sums up Higginson. Absolutely thorough, going into the minutest details of every case and of everything on which he was called upon to form or base an opinion. As one of his colleagues expressed it to me: 'He had a reason for every minutest quantity of what he put into a prescription.'

"He was one of the Honorary Consulting Surgeons to the Southern Hospital at the time I commenced my residence there in 1872. I do not think he ever once missed attending both consultation and operation every time he was summoned,—and it was at that time the rule to send out a summons to the whole staff including the honorary consulting staff for every major or likely to be major operation. He was extremely critical, and could be very witty not to say sarcastic.

"One day some accident case had been brought in which required immediate operation. There was no time for the usual complete washing process. The man was on the table, the anæsthetic was being administered, Higginson's all-seeing eyes discovered an overlooked specimen of the *pediculus capitis* crawling across the operating table. Instantly, and with dramatic effect, the little man's menacing finger descended with a swoop on the intruder, and the words, 'Gentlemen! a ver-i-table louse,' brought forth an 'audible smile' from the rest of the staff.

"Higginson did not quite appreciate at its full value Lister's newly introduced methods. We were then in the carbolic spray stage of development of the Listerian revolution in surgery. I, fresh from Edinburgh, was in charge of the spray part of the performance. Suddenly I heard Higginson's quiet accents :—'Gentlemen! We seem to be doing this operation through a Scotch Mist'!

"He was no boaster. He never, if it could be avoided, mentioned the syringe he had invented, an instrument practically unknown outside Liverpool at that time. Once only I heard him say 'the syringe which is associated with my name.' He never received a penny for his invention, the use of which quickly became general throughout England. The form in which he originally introduced it was quite cylindrical. There were other very interesting scientific, astronomical, and horological inventions for demonstration or teaching purposes,—beautifully constructed.

"He was a loyal and staunch friend to myself and others. By the way, his wife was a sister of the well-known Harriet Martineau whose distinguished career and great ability were a noticeable feature in the literary and social life of the England of that day.

(Signed) HENRY HARVEY, M.B."

In addition to the acknowledgments I have already made I should like to express my indebtedness to Mr. Naldrett, Secretary to the Liverpool Royal Southern Hospital, for Alfred Higginson's photograph; to Dr. C. J. Macalister for his help in engaging the interest of Dr. Harvey; to Mr. H. E. Powell, our Librarian, for assistance in tracing Higginson's publications; and to the officials of the General Medical Council, whose efficiency has enabled us to rescue a historical figure from what seemed like impending oblivion.

Finally, I must express my thanks to Sir Henry S. Wellcome and Mr. Malcolm for their kindness in arranging an illustrative exhibition of clysters and enema syringes from the rich collection in the Wellcome Museum.

Section of Physical Medicine.

[*January* 15, 1932.]

Some Meteorological Factors which may affect Health.

By G. C. Simpson, C.B., F.R.S.

Whatever may be the opinion of medical men on the interaction between weather and health, the man in the street has very hazy ideas on the subject, and it is chiefly with these that I shall deal. My remarks therefore will deal less with the ordinary opinions of medical men than with the medical opinions of ordinary men.

It is a normal human characteristic to seek a cause for every effect. When we fail to find a cause amongst familiar things, we readily assign the cause to something with which we are not familiar; for if we know nothing about a thing we can, without outraging our common sense, ascribe to it properties which it may or may not possess.

There are several factors in meteorology for example, atmospheric electricity, ozone, radio-activity, ultra-violet light, etc., about which the ordinary man has heard, but about which he knows little, and which he supposes to have a direct effect on health. In considering these factors we must be particularly on our guard to recognize the extent to which this tendency to explain the familiar by the unfamiliar has been a factor in forming our opinion.

Another tendency of the untrained mind is the conviction that one's health is affected by weather because one notices that one feels off colour in certain kinds of weather. It is very difficult to determine whether one variable is the cause of another by observing coincidences. Yet we are all constantly trying to do it. If we believe that a certain relationship exists between two factors, we generally notice the occasions when they occur together, but do not think of the relationship when they occur separately. Thus we remember many coincidences but not the cases in which the relationship did not hold.

When one has two such marked variables as weather and health it is easy to convince oneself that the two are related. The only way, however, to be certain of such connections is by the well-known mathematical device of the correlation coefficient. Records must be made at regular intervals—if possible by independent observers—of the two factors to be examined and the magnitude of each at every observation expressed as a numerical quantity. The correlation coefficient of the two records can then be calculated to give the true measure of their connexion. The opinion of someone who has merely his own impressions to guide him is of no value, no matter how long he has continued his observations; in fact in most such cases the longer the series of observations the less is the value of the opinion.

The danger of arguing from analogy has played a great part in medical practice in the past. For example: It did not need much intelligence to associate bad smells with bad health, and then, by analogy, to associate pleasant smells with good health, but this analogy became dangerous when the carrying of flowers and perfume boxes became the recognized way of protection from contagion. The bouquets carried by the Lord Mayor of London and His Majesty's Judges on certain ceremonial occasions to-day are an interesting reminder of this false medical analogy.

My final remark of a general nature is on the necessity of separating the physical from the psychological effect of weather on health. Most people in this country think it unhealthy to sit, still more to sleep, in a room with closed windows. As a meteorologist I think I am qualified to say that in most rooms in this country the

air is changed sufficiently often to exclude any possibility of a chemical change in the atmosphere, and as a matter of fact, large variations in the chemical constitution of air can be made without any detrimental effects. There can be no doubt that the unpleasant feeling which many people have in closed rooms is psychological rather than physical. The air in a room receives gases from many sources—from the binding of books, carpets, upholstery, etc.—which are harmless in themselves, especially in the small quantities involved, but have distinctly unpleasant odours. It is these odours which we notice and find intolerable, although they may have no direct detrimental effect. We react mentally, not physically.

Pressure.—So far as we know, the body is not affected directly by the pressure to which it is subjected. Rapid changes of pressure have unpleasant and even dangerous effects, but natural changes of barometric pressure are much too slow to be effective. There are, however, two indirect effects of pressure which might be mentioned. When the pressure changes, the quantity of oxygen in a given volume changes with it. Thus when the pressure is high the concentration of oxygen is high, while when it is low there is a deficiency of oxygen. This change in quantity of oxygen is not important at sea level where the changes of pressure are small, but by ascending mountains or descending into mines the pressure may be halved or doubled, and with it the quantity of oxygen. Distress at high altitudes, with the well-known mountain sickness, is probably entirely due to dearth of oxygen and not to the direct effect of reduced pressure.

Ellsworth Huntington has pointed out the possibility of another indirect effect of pressure on health. Some slight correlation between well-being and the height of the barometer has been observed in America. Huntington explains this by the fact that low and high barometric pressures are closely associated with the passage of cyclones and anticyclones, with their characteristic weather conditions. It is these varied weather conditions—not the pressure itself—which are the cause of the apparent correlation between barometric pressure and well-being.

Temperature.—It is not the temperature of the air but the rate of loss of heat from the body which has a physiological effect. In this loss of heat the air temperature, in the absence of wind, plays a surprisingly small part, and very little clothing is sufficient to protect the body from undue loss of heat, no matter how cold the air may be I remember, when in the Antarctic, being called out of bed to observe an unusual display of aurora. With an ordinary overcoat over my pyjamas I went out in the open, and stayed there for well over an hour with the temperature more than 40° below zero Fahrenheit, with no ill effects because there was no wind. On the other hand, one can be "chilled to the bone" in the artificial wind experienced in an open motor car with the temperature well above freezing point.

A little known effect of great cold is that in the neighbourhood of − 50° F. it is extremely difficult to do strenuous work involving deep breathing, for the cold air entering the lungs produces considerable pain. Running is quite impossible in such circumstances.

Humidity.—Water vapour is one of the few constituents of the atmosphere which varies from time to time and from place to place. It has, however, a characteristic which no other constituent possesses, namely, that its quantity is strictly limited by the actual temperature of the air.

Hence arise the two methods of specifying the humidity of the atmosphere: (*a*) the absolute humidity, which expresses the quantity of water vapour present, and (*b*) the relative humidity, which is the ratio of the amount of water present to the maximum which could be present with the existing temperature. Both these aspects of humidity play a part in the mechanism of a living body, but the relative humidity is the more important. The absolute humidity is only important in connection with breathing. The air exhaled in breathing leaves the lungs completely saturated. The same quantity of water leaves the body with every

breath, no matter what may be the humidity of the atmosphere, but the quantity of water taken in with each ingoing breath depends on the absolute humidity of the air. The difference in the quantity of water taken in and sent out in breathing has to be drawn from the water already in the body. Now at very low temperature the air can hold only insignificant quantities of water even when completely saturated; therefore in cold climates breathing involves a large loss of water, hence the well-known thirst experienced by men sledging in polar regions.

The importance of relative humidity is chiefly due to the fact that the rate of evaporation from the skin largely depends upon it. It must not be thought, however, that there is no evaporation from the skin in completely saturated air, as is so frequently stated.

At all air temperatures below blood heat the surface of the skin is to some extent warmer than the surrounding air; thus even if the air is saturated, it is warmed up when it comes in contact with the skin and so can take in more moisture. An interesting example of this effect can be seen when a horse "steams" after hard driving on a humid day. The saturated air surrounding the wet horse is warmed by contact and takes in more moisture. As this warmed air rises it mixes with the surrounding air, cools, and deposits its excess water as a visible cloud.

The cooling effect of dry air is known to everyone. The temperature recorded by a wet bulb thermometer is a much better measure of the unpleasantness of a warm climate than the dry bulb temperature which is usually reported.

The temperature of the skin can never be below the temperature of the wet bulb, while it may be many degrees below that of the dry bulb, thermometer. For this reason the wet bulb thermometer is a useful danger-signal for heat-stroke, and it has been the practice of the Indian Meteorological Department during recent years to warn all military stations when it is anticipated that the wet bulb temperature will reach 80° F. in the course of the day, so that all unnecessary exercises may be stopped.

Anyone who has been in India during the hot weather will be familiar with the "tattie"; this is a screen of rushes placed over an open doorway through which a current of air passes into the house. A servant keeps the tattie wet by frequently pouring water over it. The air passing through the tattie is cooled by evaporation, bringing a welcome lowering of the temperature indoors. In a recent paper Dr. C. W. B. Normand, the present Director-General of Indian Observatories, has brought out the unexpected fact that although one can lower the temperature of the dry bulb thermometer by the use of a tattie one cannot lower the temperature of the wet bulb. Thus the wet bulb temperature inside a house with a tattie will be the same as outside.

Wind.—Of all direct effects of the weather on health and well-being, I believe that of wind to be by far the most important. Though wind does not alter the physical characteristics of the air, it materially affects the action of these characteristics on bodies exposed to it. The temperature of air remains the same whether it is still or in motion, and it is not the temperature of the air but the rate of loss of heat from the body, which is physiologically important. This depends on the motion of the air as well as on its temperature. Similarly, much of the cooling of the body is due to surface evaporation, and the rate of evaporation with any given relative humidity increases with the wind velocity. Thus whether air is moist or dry, if its temperature is below blood-heat, the effect of setting it in motion is to increase the rate of loss of heat from the body. When one is in good health, loss of heat sets up pleasant and health-giving reactions and stimulates one to take exercise which also is health giving. This brings me to the consideration of what makes one place bracing and another relaxing. I remember discussing some years ago with other meteorologists, the question why of two seaside resorts within sight of one another, one was bracing and the other relaxing. They were so near together that there was no question of

difference in the ordinary meteorological factors such as temperature, humidity or wind velocity when measured by a well-exposed anemometer. We reached no decision then, but further experience has I believe given the clue. Both towns have good promenades, but while in one case the sea comes right up to the promenade there is half a mile of sand between the other promenade and the sea. If, now, one measures the wind velocity, not by a perfectly exposed anemometer thirty or forty feet above ground on a site free from all obstructions, but where the people spend most of their time, that is three or four feet above the promenade or on the sands, one would find that the wind velocity on the promenade near the sea would be appreciably higher than on that half a mile away from the sea. It is this difference in wind velocity which makes one place bracing and the other relaxing. This was brought home to me during a stay in Simla.

Simla is 7,000 ft. above sea level—that is, higher than most Alpine resorts—and it has a temperature similar to that of a town in England, yet people constantly complain that Simla is not bracing. The explanation is, I believe, that for the greater part of the year there is practically no wind in Simla.

In England there is not much difference between the temperature and humidity at the various seaside resorts, but there is a great difference in the mean wind velocity measured on the promenades, and I cannot help feeling that in this wind velocity lies the difference in their bracing qualities.

Wind can obviously cool the body only if the air temperature is below body temperature; if it is higher, wind will tend to warm the body. I say "tend" because if the air is dry as well as warm the wind will increase evaporation and so tend to cool the body. These two tendencies act in opposite directions, and it is important to know which is the greater, for if the warming is greater than the cooling, the body temperature will rise above blood-heat and so cause death; while if the cooling is greater than the warming, life may exist in air temperatures well above blood-heat. The answer will obviously depend on the temperature, the wind velocity and the relative humidity. Dr. Normand has studied this problem and obtained some interesting results. He finds that in calm air the normal human body can support 100° F. if the relative humidity is less than 90%, 120° F. if it is less than 40%, and 140° F. if it is less than 15%, but that even if the air were quite dry death would occur at 128° F. if the wind velocity were 9 metres per second (20 m.p.h.), at 117° F. with a velocity of 25 m./sec. (56 m.p.h.). These velocities and temperatures are not unknown to occur together in hot desert winds, such as the simoom, about which there are many travellers' tales of death due to the hot wind.

An important effect of wind, especially for town dwellers, is its influence on atmospheric pollution. Wind has two aspects with regard to atmospheric pollution: in the first place its velocity is important because when it is high the wind distributes pollutions over a large volume of air, and so reduces the concentration of the obnoxious constituents; in the second place the direction is important because wind from some directions brings contaminated air, and from others pure air to the place of observation. I am not prepared to estimate the ill effects on health of the pollution of the atmosphere from an ordinary manufacturing town; but there is evidence that industrial towns *per se* are not unhealthy. There can however be little doubt that the psychological effect of pollution, with its dull skies and unpleasant odours, is depressing.

With regard to the health-giving properties of our seaside resorts. How far it is the medical opinion I cannot say, but the popular opinion is that sea air contains health-giving qualities not present in ordinary air. By the principle of explaining the unknown by the unfamiliar, the health-giving property of sea-air is often ascribed to ozone. However, Lord Rayleigh, in 1918, with the most delicate instrument then available—the spectroscope—could detect no ozone in the lower atmosphere. Recent and still more delicate observations have been able to detect a trace of ozone

in the lower atmosphere, but so small (3 milligrams in 100 kilograms) that it could not possibly affect health, and there is reason to believe that land air, especially polluted air from towns, has more ozone than sea air.

I suggest that the health-giving properties of seaside resorts are due on the physical side to the "bracing" effect of the relatively high wind velocities, and on the psychological side to the absence of familiar town odours and the presence of sea odours. Town dwellers detect the unfamiliar sea odours at once, and have little difficulty in convincing themselves that the sea air does them good.

Fog.—Fogs and mists have always been supposed unhealthy. From a physical point of view it is difficult to see what effect a clean fog can have on the body. The air is no different after a fog has formed in it from what it was before the fog formed—except possibly it is a fraction of a degree colder. Every breath of air taken into the lungs contains just the same amount of water, both before and after the formation of the fog. If clean fog is detrimental to health it must be through its psychological effect. The case is different where the fog forms in a polluted atmosphere. A fog has exactly the opposite effect to wind on pollution. While wind rapidly dissipates pollution the meteorological conditions which accompany fog all tend to concentrate it. In most fogs there is practically no movement of air either vertically or horizontally, so that the pollution remains where it is emitted. The high fogs which turn day into night in London are due to the holding of all the smoke just above the house tops; this smoke loaded with the water of the fog, is impenetrable to light. In such conditions the pollution near the surface can rise to dangerous amounts and even the layman can see that these fogs must be unhealthy.

Radiation.—There are three effects of radiation on the human body: (*a*) heating, (*b*) chemical and (*c*) psychological.

Heating.—Electro-magnetic radiation can take various forms; these are, broadly speaking, wireless rays, infra-red rays, light rays, ultra-violet rays, X-rays, gamma-rays and ultra-penetrating (cosmic) rays. All these rays are electro-magnetic disturbances which carry energy and they differ from each other only in that they have different wave lengths.

When an electro-magnetic wave is absorbed its energy appears as heat *where the absorption takes place.* This is true of all kinds of radiation and the ultimate method of measuring the intensity of any radiation is to absorb it and measure the heat produced.

In meteorology we need only consider two ranges of wave length: first the wave lengths associated with visible light and secondly the somewhat longer wave lengths called infra-red rays.

The radiation sent out by the sun consists of all wave lengths, but the proportion of short waves is so great that we are justified in considering that solar radiation consists only of short wave lengths. When these waves are absorbed they produce heat; but their outstanding characteristic is that within the visible range bodies absorb the various wave lengths very differently. White substances reflect all these rays, coloured bodies reflect some and absorb others, while black bodies absorb them all, thus with sunlight the colour of the bodies on which they fall is the predominating factor which determines their heating effect.

The second range of wave lengths—the infra-red—are emitted by all bodies at atmospheric temperature. The chief characteristic of these rays is that colour has no influence on them and they are absorbed equally by all bodies. In fact, to these rays all bodies are black—snow and tar, white skin and black skin absorb them equally.

Most of us are able without difficulty to visualize the absorption and emission of radiation by solid and liquid bodies, but the laws of radiation of gases give us more difficulty. As the radiation from the atmosphere is very important to the human body it is necessary that we should understand it.

Perfectly dry air neither absorbs nor radiates; if the air were completely dry it would play no part in the exchange of heat by radiation. On the other hand, water vapour absorbs and radiates long wave lengths very efficiently. Except for a slight absorption in the so-called rain bands water vapour is transparent to visual light, but it absorbs a very large proportion of the long wave length radiation emitted by bodies at earth temperatures. It is, therefore, able to emit these long wave lengths, and if the absolute humidity is high, as it frequently is in tropical climates, the water vapour in the air acts like a black body and emits nearly the full radiation of a black body at its own temperature. We shall see later that this atmospheric radiation plays an important part in health.

The practical application of these laws of heat radiation to health is most important in connection with clothing in the tropics. Grabham has made some interesting observations in the Sudan on the absorption of solar radiation by different fabrics. A number of different types of cloth were exposed to the sun on a support 18 inches above the ground; a thermometer was inserted in each so that its bulb was covered by a single thickness of cloth and separated from the support by at least five thicknesses of the same cloth. The following table shows the maximum temperature reached by each kind of cloth on a day when the maximum air temperature in the shade only reached 109° F.

TABLE I.

	Temperature ° F.	Excess over maximum air temperature.
Thin black lining	194	85
Black serge	194	85
Blue zerak[1]	183	74
Thick khaki drill	176	67
Thin khaki drill	167	58
White duck	149	40
Thick white drill	145	36
Maximum air temperature	109	—

[1] A dark blue Manchester cotton cloth dyed in Egypt.

This table shows clearly the importance of the colour of clothes if one wishes to remain cool when exposed to solar radiation, but it must be clearly realized that the effect is due to the selective absorption of short wave radiation. If there is no short wave radiation then all cloths absorb equally and all tend to take the temperature of the air. White evening dress for men as well as women is common in the tropics; but as it is not worn until after sunset when there is no short wave radiation, the colour of one's clothes is immaterial. A white drill dinner jacket is pleasanter to wear on a hot night than an ordinary black one, but this is merely because it is light in weight and has no lining. Out of direct daylight the colour of clothes does not matter; so that in order to keep cool one does not require a dress of light colour but one of open texture which allows free circulation of air to the skin.

There is always considerable discussion among ordinary men who have lived in the tropics regarding the true cause of sunstroke. Their chief difficulty is in explaining why one can go about in England even at midsummer, when the altitude of the sun is 60° or more, without a hat, while in India one would never think of going out of doors, even soon after sunrise, without wearing a solar topee. Also it is said that in North Australia white men do not wear topees and yet sun-stroke seldom occurs. Naturally the usual explanation includes ultra-violet light, but why there should be more ultra-violet light in India than in England or Australia is not considered—the mere use of the word is sufficient. Is there no more reasonable explanation? I may state at once that the heating effect of solar radiation, with the sun at a given altitude, is practically the same in all parts of the world. What little difference there is is in favour of higher latitude where there is less dust and water vapour in the atmosphere than in the tropics. We may accept, I think, that if any part of

the body, especially the head, becomes warmed to a higher temperature than blood-heat, the heat-regulating mechanism of the body becomes deranged and there is at once a source of danger. Direct solar radiation is only one factor which raises the body temperature, there is in addition the infra-red radiation from the ground and from the air. In relatively cool conditions these are not large, but as the air temperature rises and the amount of water vapour in the air increases, the infra-red radiation from the ground and the air increases very rapidly until at tropical temperatures the intensity of the infra-red radiation approaches that of the sun itself. On the other hand, the high air temperature and the high humidity reduce the natural cooling of the body by convection and evaporation. Thus the high temperature and humidity act twice: once in increasing the infra-red radiation and then again in decreasing the cooling. Finally, in India there is little air movement during the hot weather. The conditions are therefore extremely favourable for the attainment of high local body temperatures, especially on the head if it is uncovered.

If it is true that in parts of the world nearer to the equator than India little or no sun-stroke occurs, then, I think, it will be found that in every case the air temperature is lower, or the air drier, or the wind velocity greater than it is in India when the sun attains the same altitude. Further, in Australia white men have to do hard manual work in the open and in consequence become acclimatized and therefore can tolerate conditions which would prostrate the office-working white man in India.

In connection with sunstroke, I came across in India one of the best examples of false reasoning from analogy that I have ever met. I was consulted by the military authorities who had been advised to have all tunics lined with a red fabric "in order to keep out ultra-violet light." The reasoning which led up to this advice seems to have been something like this: A photographer uses red glass to keep out short wave radiation, therefore, if we line tunics with red fabric we shall keep out ultra-violet light. In my reply I pointed out that a photographer uses a red glass because he has to let some light through, otherwise he would not be able to see what he is doing. If his object were merely to keep out light he would use an opaque screen and not a red one, therefore there was no particular virtue in the red colour. I added that, as very little radiation of any wave length, especially ultra-violet, would penetrate a khaki tunic, the colour of the lining would be of no importance whatever.

Chemical effect of radiation.—This subject has recently been much in the public eye, especially in connection with ultra-violet treatment. Let us consider the purely physical aspect of the question: Radiation of long wave length has little or no chemical action, but as we examine shorter and shorter wave lengths we find that chemical action first appears with red rays, becomes appreciable with orange, and of importance with green. As we proceed to examine still shorter waves, through the blue and violet to the ultra-violet, we find the chemical activity of radiation steadily increasing; the reason for this is now well understood in terms of quanta. Now anything which induces chemical action in human tissues may produce good or harmful results. In fact radiation acts like a drug and each wave length corresponds to a different drug.

That is all that, as a physicist, I am justified in saying; as a meteorologist I might add that owing to the layer of ozone in the upper atmosphere, there is very little ultra-violet light in solar radiation, but as an ordinary man given to arguing from analogy, I should like to ask my medical friends one or two questions. Nature always makes use of any useful property of the natural world and I notice that she makes great use of the chemical properties of radiation in building up the vegetable kingdom, but when it comes to the animal kingdom it appears to me that she has found it necessary to protect the surface of the human animal from the more active chemical rays by pigmentation induced by the chemical rays themselves. What I cannot understand is why it is considered good for us to get all the ultra-violet

light we can out of the sunshine when Nature herself says that it is not good for us. I am prepared to take doses of real ultra-violet light prescribed for me in regular doses by a qualified physician, but I am not prepared to go sun bathing, or ski-ing in the snow without clothes, because certain doctors, arguing from analogy, I believe, say that one must have all the ultra-violet light one can get. One does not prescribe castor oil in that way.

Psychological effect.—I know that beneficial use can be made of the heating and of the chemical properties of sunshine; but it is my strong belief that the greatest effect of radiation on the well-being of man, whether in health or in sickness, is psychological. To be able to sit out in the warm sun on a cold day gives pleasure and a feeling of well-being which has little to do with the actual heat received (that could be got indoors from a fire) or with the chemical effect on the blood (that could be got from drugs). The satisfaction is purely mental and so reacts throughout the body.

This satisfaction, however, depends largely on the dull days which have preceded the sunny ones. After two or three months of continuous sunny weather at Simla it gave me a strange feeling when I came down one morning to find a dull day. It gave me almost the same sort of mental stimulus which one has in England on a sunny day after weeks of dull weather.

Atmospheric electricity.—It is impossible here to deal with the large subject of atmospheric electricity in anything like a complete way; I therefore confine myself to general considerations. The electrical state of the atmosphere, whether in normal fine weather or in the abnormal conditions of a thunderstorm, can only act through three physical agents, namely: (*a*) the electrical field; (*b*) free electricity, and (*c*) radio-activity.

The electrical field.—There is always an electrical field in the atmosphere in all parts of the world; its intensity is measured by the difference of electrical potential at two points vertically above one another and one metre apart—this is called the potential gradient.

Measured thus, it is found that in fine clear weather the potential gradient is about 200 volts per metre. In particularly clear air it may fall to 100 volts per metre and in the polluted air near large cities it rises to 300 volts per metre. This means that the electrical potential at the height of a man's head is generally about 300 volts above the potential of the earth—that is, a difference of potential greater than that used in our electric lighting circuits. As soon as the weather becomes disturbed, the electrical field changes. With rain of the non-thunderstorm type the sign of the field is usually reversed and instead of positive potential gradient we find negative potential gradient, but of the same order of magnitude, i.e., a few hundred volts per metre. During thunderstorms, however, electrical fields of an entirely different order of magnitude come into play. Fields of a hundred thousand volts per metre are quite common over large areas during thunderstorms. One's natural reaction to such statements is to wonder how any of us can exist in such strong electrical fields, seeing that 200-volt lighting circuits can give fatal shocks.

The answer is that the body is no more aware of an electrical field than of the gravitational field of force; it is only when that field of force sets up electrical currents which pass through the body that physiological effects come into play. Under normal conditions the air is such a bad conductor of electricity that appreciable currents cannot pass through it. In consequence, when we walk about in the open air in fine weather, and the electrical field tries to pass a current through our bodies with a force of several hundred volts, it is quite impotent to do so because the air cannot receive the current from the body and no flow of electricity takes place. For simplicity I have over-stated the case in the last statement, for there is in fact a small current through our bodies even with the normal electrical fields; but it is so small that I am justified in calling it zero. When,

however, the field reaches its highest values in the neighbourhood of thunderstorms, there is sometimes a discharge of the nature of Saint Elmo's fires from bodies exposed in the open; we need not, however, here discuss such abnormal and infrequent discharges. The point I wish to make is that whether the field is the normal one of fine weather or the high field of a thunderstorm, there is no direct physiological effect on bodies exposed to it, because the field does not set up electrical currents in the body itself.

Furthermore, the abnormal electrical effects do not appear until the thunderstorm has developed and then they are limited to its immediate neighbourhood. Five miles away from a thunderstorm the electrical conditions are generally normal. Most people consider that the atmosphere becomes more and more electrical until a thunderstorm results. The facts are quite the reverse. There are no abnormal electrical conditions until the rain-storm has developed; then, and only then, does the rain produce the electrical effects associated with a thunderstorm. It is therefore not correct to talk about "thundery conditions" until a thunderstorm has developed and come quite near to the place of observation.

I now pass on to consider the free electricity in the air. The normal molecule of air is electrically neutral, but there are always a few molecules which have an electrical charge: this charge may be either positive or negative. Charged molecules are called ions: positive ions if they carry a positive charge and negative ions if they carry a negative charge. There are two main types of ions in the atmosphere, small ions and large ions. The former appear to be single molecules of air which, after becoming charged, collect a number of water molecules about them, so that a small ion is a charged molecule of air enclosed in a bunch of water molecules. The large ions appear to be of a different nature. There are always in the atmosphere a large number of hygroscopic nuclei which are supposed to be mainly crystals of sea-salt, or hygroscopic bodies produced by combustion. Some of these nuclei capture small ions, and thus become charged themselves. Nuclei so charged are the large ions.

Similar ions are found in chemical solutions, where they play a large part in chemical and electrical reactions; it is therefore natural to ask whether the ions in the atmosphere may not play some part in physiological processes.

A priori the physicist is inclined to reject any such idea, for several reasons. First, the number of ions in the atmosphere is so small—one in a thousand million million neutral molecules—that it is inconceivable that they can play any important part in bodily functions; secondly, in the process of breathing the air passes through damp narrow passages where the electrical charge on the ion is lost and only neutral molecules enter the lungs; thirdly, there are practically the same number of positive and negative ions and therefore so far as any electrical effect is concerned they neutralize one another.

A considerable amount of work has been done on this aspect of ionized air and the anticipations just stated have been confirmed. Dorno has measured the natural ionization of the air at Davos for many years and can find no direct effect on health. More recently Dr. Dessauer and his collaborators in Frankfurt have made many experiments on artificially ionized air and find that while large doses of ions all of one sign have certain pathological effects, no effect of large quantities of oppositely charged ions can be detected; from which it is clear that no effect from the much smaller number of naturally formed ions can be expected.

Soon after the discovery of radio-activity it was found that there is always a measurable quantity of radio-active matter in the atmosphere. At once all sorts of medicinal properties were ascribed to the radio-activity of the air. Further knowledge of the action of radio-active substances on the living body have, however, shown that the quantities of radio-active matter in the atmosphere are absolutely insignificant from the point of view of health.

Having examined all the physical aspects of atmospheric electricity and found none of physiological importance, one may ask why thunderstorms have such marked

effects on certain people. This question might be met with another: Why does a thunderstorm turn milk sour? The answer is substantially the same in both cases. It is not the electricity of the thunderstorm which causes milk to go sour and people to have headaches, but the accompanying meteorological conditions, for in this country at least thunderstorms generally occur with hot and humid conditions which are favourable to the growth of bacteria and are unpleasant to most people. If to these general depressing conditions we add the nervous strain due to physical fear of thunder and lightning I think we have sufficient explanation of the discomfort of some people in thundery conditions without the need to invoke any effect of electricity.

Dr. C. W. BUCKLEY called attention to the observations of Tyler on the climate of Shanghai communicated to the Balneological and Climatological Society 25 years ago. He got a body of picked observers to register their subjective impressions of the bracingness or otherwise of the weather for a month on a scale of degrees from one to ten. These records were then compared with the records of the wet and dry bulb thermometer but no definite relation was found. He (the speaker) however, analysed the figures further and found that the curve of the observations made coincided almost exactly with that of the absolute humidity. The reason for this was made clear by Dr. Simpson's observations that low absolute humidity, by leading to greater evaporation, and cooling was more bracing. He also called attention to Sir Herman Weber's observation that in respect of certain climatic characters, notably bracingness, an altitude of 1,000 feet in this country was equivalent to one of 3,000 to 4,000 feet in the Alps and 5,000 or 6,000 feet in the Himalayas, which the lecturer's observations indicated was due in some degree to wind velocity and consequent evaporation. The lecturer held the view that the influence of weather changes on the human organism was largely if not entirely psychical, apart from the effects referred to, but this failed to explain the sensitiveness of rheumatic patients to certain weather conditions, especially high winds, of which they were conscious even when indoors, and the anticipation of weather changes manifested by the lower animals. Just as pharmacologists had in the past asserted that certain drugs could not possibly have the curative action with which they were credited, but had later been obliged to admit that empiricism had been justified in the light of further research, so he believed that further knowledge in meteorology would throw a light on these effects of weather which were too universally believed in to be set aside lightly.

Dr. J. KINGSTON BARTON said that in the *Morning Post* of January 11 there had been published an interesting graph showing the great variation in temperatures observed at Kew during the previous month. He (the speaker) had correlated these changes with the barometer changes for the same period and also with certain figures from the weekly reports of the Registrar-General most likely to demonstrate effects of weather—namely, those of the deaths from "Old Age," from "Respiratory Diseases, excluding Tuberculosis," and from "Cerebral Hæmorrhage." [Dr. Kingston Barton exhibited on the epidiascope the composite charts which he had devised.]

Section of Odontology.

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Preliminary Investigation of the Influence of Raw[1] Milk on Teeth and Lymphoid Tissue.

By Evelyn Sprawson, M.C., L.R.C.P., M.R.C.S., L.D.S.

The problem of dental caries is beset with many difficulties, and many factors seem to be involved. One feels, therefore, that clinical observations which seem to point to some measure of prevention should be recorded. Clinical facts are incontrovertible, and the coincidence of their occurrence in association with other circumstances must be taken into account. Coincidence it is true has a long arm, but it is not of unlimited length. The decision whether the clinical facts recorded are caused by the associated circumstances cannot be a personal one, but must ultimately depend on the experience and observation of many.

Observations Leading to the Investigation.

The observations herein recorded seem to show that raw milk, whether such as Nature provides for the purpose, or that of cows, has a profound influence on the development and calcification of the teeth of man, in that it confers on them some immunity to dental caries.

I have had under my dental supervision since 1908, collections of children in residential institutions. These consist of: at A, some 1,100 girls; at B, some 750 boys (A and B are under the same organization); and at C, unconnected with A or B, some 200 boys and 100 girls. Before admission these children do not as a rule receive that home care which is desirable, principally on account of poverty in A and B; in C conditions are probably better, but in many cases antecedent conditions are far from ideal. Their ages range from 3 or 4 years at A and B, and 7 years at C, to 16 years at all of them.

Prior to 1910 I tabulated [1] the incidence of dental caries in 2,000 of these children at different ages, taking the average of several hundreds at each institution. The incidence of caries per cent. and per 400 first permanent molars per hundred children, was as follows:—

At ages 10, 11 and 12 years taken together,

In A, 78% had dental caries, 148 first permanent molars were carious.
In B, 86% „ „ „ 183 „ „ „ „ „
In C { 100% girls had dental caries, 300 first permanent molars were carious.
{ 95% boys „ „ „ 227 „ „ „ „ „

Increased ages sometimes showed a higher incidence but those details are not relevant. There is no record of their incidence of caries at earlier ages, but the following figures are informative and are from statistics compiled for the Salop County Council in 1914 [2] in which it is recorded that of 3,794 children aged 5 years, 95 % had carious teeth. The average number of carious teeth per head was 6·8; 1,017 had 10 or more carious teeth each.

At B, from 1925 onwards, the boys have had a daily ration of one pint of raw milk. At the time I did not know this, but a change in the nature of my work, first noticed about 1928 and increasingly since, prompted me to make inquiries as to alterations

[1] By raw milk is meant milk which has at no time been heated to more than a few degrees above body temperature.

in their diet or environment which might be responsible for it. This change was a remarkable diminution in the number of "return" cases, i.e., once they had had dental treatment there was but little subsequent treatment for dental caries needed during their stay at the institution.

At first this was attributed to the calcifying vitamin D, which is contained in small amount in milk, but experience at C, where cod-liver oil (with its larger vitamin D content) is freely used, did not confirm this, as here there is a great deal of "return" work. Milk at C is delivered pasteurized and is not a regular item in the children's diet throughout their entire stay, but all the younger ones, and such older ones as are subnormal in physique or otherwise, are given extra milk.

There are at C fifteen of these older children who are subnormal in physique, they have had 12 oz. of pasteurized milk daily during school term, since admission; the details of their increase of caries are as in Table I.

TABLE I.

Children having 12 *oz. of pasteurized milk daily during school term since admission.*

Number of children	Average age at admission	Average age now	Increase of caries since admission
15	$8\frac{5}{12}$ years	$14\frac{5}{12}$ years	67 cavities in 14 children

20 of these cavities are in the second permanent molars, which erupt during the thirteenth year. The fifteenth child was the only one with no caries at admission and has none now.

There are also at C, another eight of these older children who, in addition to 12 oz. of pasteurized milk have had cod-liver oil (Evans, Lescher and Webb's "Special") daily during school term since admission: the details of their increase of caries are as in Table II.

TABLE II.

Children having cod-liver oil and 12 *oz. pasteurized milk daily during school term since admission.*

Number of children	Average age at admission	Average age now	Increase of caries since admission
8	$9\frac{3}{12}$	$15\frac{7}{12}$	34 cavities in 6 children (18 of these are in one child)

14 of these cavities are in the second permanent molars, which erupt during the thirteenth year.

The two children with no increase were the only ones with no caries in permanent teeth at admission.

The three children in Tables I and II who have no increase in caries are the only ones of these twenty-three children who were caries-free on admission,[1] at which time they were aged $7\frac{1}{12}$, 8 and $10\frac{9}{12}$ years; they are now aged respectively, $10\frac{9}{12}$, $15\frac{1}{12}$ and $14\frac{5}{12}$ years; it would seem, therefore, by comparison that whatever determined their freedom from caries was something that happened before they came to C, and so quite early in their lives.

It might be thought that a high degree of calcification of the enamel, such as is said to be caused by vitamin D, would be a protection against dental caries, but clinical and histological examination of many teeth under normal and pathological conditions amply demonstrates that a high degree of calcification is in itself no protection, as in the highest degrees of calcification I have seen, dental caries was present.

There is also the evidence of J. W. Field [3] of the Malayan Medical Service concerning people who work with large areas of their bodies exposed to direct sunlight with the resultant formation of vitamin D, by its action on the ergosterol content of the skin. He compares the incidence of caries in Chinese and Tamil

1 All three were free with regard to the permanent teeth, but one of the children in Table II had a carious deciduous first molar.

coolies working in the same district, "most Chinese coolies work stripped to the waist for eight or more hours a day exposed to powerful solar irradiation," yet, on comparing two groups of coolies taken without selection, he found:—

	Number with caries	Number of carious teeth
Chinese per 100	49	163
Tamils per 100	6	7

Concerning this also, F. William Fox [4] of the Biochemical Department, South African Institute for Medical Research, writes: "Bright sunlight is available almost every day of the year to irradiate our skin and the food we eat. It is difficult to believe that there can be any deficiency of vitamin D especially when we find that rickets is almost unheard of. In spite of this, however, dental caries is unfortunately only too common; in the opinion of some authorities the incidence is at least as great as, if not greater than, in England. Moreover it is not confined to the European. Although the fine teeth of the Bantu are proverbial, investigation has shown that dental caries is by no means uncommon, even among the native children, whose life in the kraal might almost be described as a perpetual sun bath."

Again, G. Friel and Professor J. C. Middleton Shaw [5] of the Department of Dentistry, University of Witwatersrand, Johannesburg, South Africa, write: "In Johannesburg we are favoured with a large amount of sunlight and, owing to our altitude the intensity of the ultra-violet rays is greater than at lower altitudes. The actual figures given by Osborn [6] are that, of the active rays given off by the sun, provided there are no clouds, 26 % reach the earth in England, 43 reach the earth on the high veldt, e.g., Johannesburg. But in addition, the sun only shines 29 % of the total possible in England as against 73 % in Johannesburg. Taken all together, Osborn estimates that if the actinic rays in England are estimated by one, we in Johannesburg have ten." Yet they record that of 600 white children aged from 6 to 16 years, 93·33 % had carious teeth, the average being 4·82 carious teeth each.

A. T. Till [7] writing of native children, says they "live in a country that is rich in sunshine, and as they wear very little clothing the rays from the sun play directly upon their bodies," and records that of all ages up to 15 years, taken together, 68·9 % have caries.

Even if the extreme of difference in the incidence of caries, as recorded by these last three observers, between white and native children in South Africa be ascribed wholly to exposure to sunlight, it still leaves the incidence at approximately 69 % in spite of the small difference in the age range. In view of what follows one cannot but think that the 24 % reduction in the incidence of caries in these Swazi children is due rather to their longer period of maternal feeding, but that similar food was not continued long enough for their efficient protection.

THE INVESTIGATION, THREE CONTROLS, FINDINGS AND REMARKS THEREON.

I had therefore to reconsider my views concerning the relative absence of "return" cases at B, and arrived at the following tentative hypothesis: That possibly such balance of vitamin content as is in raw milk, in conjunction with the calcium in it (which may be in a particularly suitable form), may be the cause of the diminution of "return" work; these acting, not so much by causing a higher degree of calcification as by providing simultaneously calcium salts of a more stable, resistant, assimilable or ideal form for deposition in the forming enamel.

Now it is a fact that a very high percentage of the incidence of caries occurs in those deciduous and permanent teeth the enamel of which is actively laid down, and in some cases fully calcified during the first four or five years of life, so that it is highly desirable that a supply of suitable calcium salts and vitamin balance and efficiency should be available at that period for the ideal deposition of the forming enamel. It is also true, except in such teeth as are protected

by their non-retentive shape, that the incidence of caries is, generally speaking, progressively less in the teeth, deciduous and permanent, in the order of their eruption: and it is also true that during the earliest portion of this period, "hand feeding" and faulty dietary are most apt to occur. Progressively with its age the child exercises choice in its foods, the rate of enamel deposition is slowed down, and the incidence of caries in the teeth then forming becomes less. These facts, coupled with the observations at B, prompted an inquiry concerning the possible effects of a daily ration of raw milk *commenced during the first twelve months of life.*

That age was selected because relatively few children are now breast-fed, and under normal conditions raw milk is the physiological food of a child during that period. Moreover, it is a period previous to the eruption of the deciduous molars (in which the incidence of caries is very high), and also when the first permanent molars are forming, and in these the incidence of caries is higher than in any other of the permanent teeth.

I thought that if diet could influence the structure of enamel so as to make teeth more resistant to caries, it would have to effect that influence while they were developing, and that but little change could be effected as they approached completion: though, of course, a tooth is not completed in form till a few years after eruption, and I have shown [8] that the calcification of enamel is continued for a still longer period after eruption. One regarded a tooth as one regarded the child as a whole, in that faults produced in its construction by trophic errors were probably permanent, or only likely to be slightly affected by conditions of trophic perfection introduced as development approached completion.

I therefore investigated the incidence of caries in children who conformed to these conditions, but as the deciduous dentition is not fully erupted till about the end of the second year, children under 2 years of age were not taken into account.

At A, and two different branches of the same organization, I was able to find three groups of such children as follows:—

Group I.—Four children of an average of $5\frac{9}{12}$ years, who commenced raw milk at an average age of 9 months.

Group II.—Nine children of an average age of $3\frac{8}{12}$ years, who commenced raw milk at an average age of 2 months.

Group III.—Twenty-four children of an average age of $3\frac{3}{12}$ years, who commenced raw milk at an average age of 5 months.

In addition, I have obtained information from competent observers of three other cases of an average age of 9 years, so that I am now tabulating forty cases of an average age of 4 years.

Admittedly this number is small to generalize on, but even so, the findings are remarkable, as in no case was there a trace of caries: two children had "dead" maxillary first incisors, caused by trauma, one of these had been removed; another had lost a premolar for orthodontic reasons; apart from these none had been removed. Details are as in Table III.

TABLE III.

Commencing raw milk before 12 *months of age. Present ages range from* $2\frac{2}{12}$ *to* $7\frac{8}{12}$ *years in thirty-nine instances, the fortieth is aged* $19\frac{8}{12}$ *years.*

	Number of children	Average age at commencement	Average age now	Incidence of caries
Group I ...	... 4	... 9 months	... $5\frac{9}{12}$ years	... 0
Group II ...	... 9	... 2 „	... $3\frac{8}{12}$ „	... 0
Group III ...	... 24	... 5 „	... $3\frac{3}{12}$ „	... 0
Cases not seen by myself	... 3	... 2 „	... 9 „	... 0
Totals	40	... $4\frac{1}{2}$ „	... 4 „	... 0

In Groups I and III there are, in addition to the children already tabulated, 33 others, who similarly receive a daily ration of raw milk, but were over 12 months old when they commenced it; their average age at commencement was $1\frac{9}{12}$ years, and the latest age at which any of these commenced it was $2\frac{7}{12}$ years, their average age is now 4 years.

One expected to find some carious teeth in these children, notably so in the deciduous first molars, which erupt at the age of 12 months, but 26 of them had no trace of caries; the remaining seven had 14 carious teeth, eight of which were deciduous first molars and two incisors, which erupt still earlier; a deciduous second molar was carious in one child who did not commence raw milk till aged $2\frac{5}{12}$ years; it is highly probable that these 11 teeth were already carious before raw milk was commenced. Two other children had one and two carious deciduous second molars respectively, concerning these a note appears later. Details are as in Table IV.

TABLE IV.

Commencing raw milk after 12 *months of age and not later than* $2\frac{7}{12}$ *years, present ages range from* $2\frac{2}{12}$ *to* $8\frac{5}{12}$ *years.*

	Number of children	Average age at commencement	Average age now	Incidence of caries
Group I ...	11	... $1\frac{5}{12}$ years	... $5\frac{5}{12}$ years	... 10 teeth in 5 children
Group III ...	22	... $1\frac{10}{12}$ „	... $3\frac{3}{12}$ „	... 4 teeth in 2 children
Totals	33	... $1\frac{9}{12}$ „	... 4 „	... 14 teeth in 7 children* (11 of these teeth were erupted before raw milk was started).

It was evident, therefore, that teeth which had erupted, but were not fully formed when raw milk was commenced, were brought under its influence.

I thus had the amazing experience of examining 70 children consecutively, of whom 63 were totally free from dental caries; there were, in addition, the three cases I did not myself see, so making a total of 73 children examined.[1]

There are several points of interest concerning Groups II and III. Notable points in their dietary are: In Group II, breakfast consists of oatmeal porridge or bread and milk; at dinner they get a constant ration of milk pudding or custard. White bread only is used. In Group III breakfast consists on alternate days (except Sundays) of oatmeal porridge or quaker oats; at dinner, on five days in the week they get milk puddings, this meal always terminates with the giving of one or two "boiled" sweets. White bread only is used. Milk is delivered here twice daily from a local farm where the cows are tuberculin tested, and is given to the smaller children warmed; in the case of very young children it is diluted, sweetened, and citrated, so as to approximate the maternal secretion. It will be noted that the dental errors of refined and lodgable carbohydrate foods, both starches and sugars, and the supposed error of oatmeal are freely committed, but, in Groups II and III, and the three additional cases, in the 58 children tabulated (Tables III and IV), there is freedom from dental caries in all teeth erupted since raw milk was commenced. At A, B and C, there has always been a small weekly raw fruit ration, and some is also given to each of the different groups tabulated. Another remarkable feature is that all of the 24 children in Groups I and II and two-thirds of the 46 children in Group III were subnormal on admission; these subnormalities consisted chiefly of gastro-intestinal disturbances and rickets; both of these conditions are notorious for causing abnormalities in the structure of calcified dental tissues believed by many to favour the incidence of dental caries. All the children in Groups II and III live

[1] Incidentally I also saw 25 children under 2 years of age where Groups II and III are stationed, these too are on raw milk; there was no trace of caries in any of their teeth. There is a total of 63 children at the branch where Group III is stationed, aged up to $5\frac{2}{12}$ years, two of them have two carious teeth each, these four teeth were erupted before admission. Among the total of 17 children at the branch where Group II is stationed, aged up to 5 years, there is no incidence of dental caries.

in the open in loin-cloths, provided the weather is suitable, and rickety children only receive cod-liver oil at first as required.

In order to check further the accuracy of this hypothesis, I decided to examine children who had not come under the influence of raw milk till a later age, and to note the effect on the teeth then forming, for comparison with those which had already been in use for some years and were uninfluenced by previous similar treatment.

At B, therefore, I examined all boys admitted at under 6 years of age between 1925 (when the raw milk ration was started) and the end of 1928. Owing to faulty environment and feeding before admission, well over 50% of the boys at B are subnormal physically, and in many cases this is marked. The object of this control was to see what effect would be found in the first permanent molars, which erupt during the seventh year—this tooth being notoriously liable to become carious.

There were only 27 boys who conformed to these requirements, as B has a very floating population, and many are boarded out or sent to other training centres; in addition, one other is known to me. Of these 28, commencing raw milk at an average age of $4\frac{8}{12}$ years, and now of an average age of $8\frac{10}{12}$ years, 27 had no trace of caries in their first permanent molars or any other of their permanent teeth, two of them had extreme degrees of "hypoplasia," and two others had slight degrees; most of them had, or had had, carious deciduous teeth, up to nine, still present in one mouth, and one, admitted at $5\frac{11}{12}$ years, and now aged $10\frac{8}{12}$, had stained fissures on both mandibular permanent molars. Where the cavities in the deciduous teeth had become more or less self-cleansing from food debris, spontaneous arrest of caries had usually occurred, but not otherwise.

The 28th boy is an interesting exception. He was admitted under the organization controlling A and B, aged $\frac{8}{12}$ year, and had raw milk from then till age $3\frac{3}{12}$ years; from then, till $4\frac{10}{12}$, he was continuously in a hospital where the milk supply is delivered pasteurized, for surgical treatment for talipes varus, and did not again start raw milk till age $5\frac{3}{12}$ years, but has had it uninterruptedly since; his present age is $10\frac{5}{12}$ years; his maxillary first permanent molars were symmetrically and extensively carious on their medio-occlusal aspects—not the most usual aspect for his age, particularly as the adjacent deciduous second molars were caries free, as was also his full complement of other teeth; the enamel adjacent to the cavities has a very distinct appearance of faulty development, but not what is ordinarily understood by hypoplasia—it is opaque and yellow. I have no knowledge at what age these teeth erupted.

This control examination is of the greater value, as exposure to sun (other than what is ordinary and conventional) and the use of cod-liver oil were absent.

Details are as in Table V.

TABLE V.

Commencing raw milk before the sixth birthday anniversary, present ages range from 7 to $10\frac{9}{12}$ years.

Number of boys	Average age at commencement	Average age now	Incidence of caries in all permanent teeth
28	$4\frac{8}{12}$	$8\frac{10}{12}$	2 carious maxillary first permanent molars in 1 boy

In these boys of Table V, a curious and apparently paradoxical occurrence may not infrequently be seen; it is, that new carious cavities may appear in the unprotected deciduous teeth, while the protected permanent teeth are unaffected; to quote one case: a boy started raw milk at age $3\frac{8}{12}$ years, when first seen at age $5\frac{7}{12}$ years he had six cavities in deciduous second molars and was, at this early age, erupting his permanent molars and incisors; at about age $7\frac{9}{12}$ years he presented three new cavities in deciduous teeth, he is now aged $8\frac{5}{12}$ years, but has no trace of caries in any of his permanent teeth.

The boy discussed previously, who spent so long in hospital, presents the converse of this paradox, as his deciduous teeth (all his deciduous molars are present) were protected, but apparently his maxillary first permanent molars escaped protection, possibly his prolonged stay in hospital had something to do with this.

Thus, in the 101 children (Tables III, IV and V) who had conformed to requirements and most of whom were subnormal, presuming that the dates of eruption of their teeth were normal: of the teeth erupted since raw milk had become a part of their daily diet, five only, in three children, were carious. Of these three children: one, the boy who spent a prolonged period in hospital, has already been referred to: another, a girl in Group I, admitted at age $1\frac{6}{12}$ years, "soon [9] after . . . . . was admitted to hospital[1] and for nearly two years was in and out with whooping-cough, acute bronchitis and measles at different times"; she is now aged $6\frac{8}{12}$ years and has a small carious cavity on the occlusal aspect of a deciduous second molar. The third child, also in Group I, was admitted aged $1\frac{10}{12}$ years, is now aged $8\frac{5}{12}$ and has two carious deciduous second molars. I am assured that she had raw milk "except [9] in the very hot weather when it has to be scalded to keep it from turning sour and in the winter . . . hot . . . on going to bed and before going to school in the morning"; as she was admitted in the winter it is uncertain at what age she really commenced raw milk, whether before or after the eruption of the teeth affected, in any case she has not had it very consistently, each tooth has a very small medio-occlusal cavity; caries, however, is not active but has become spontaneously arrested.

A further control was then done at a still later age, and all boys at B, admitted before the completion of their eleventh year, and now aged $13\frac{6}{12}$ years or over, were examined. The object was to see what effect would be found in the second permanent molars, which erupt in the thirteenth year, when raw milk was commenced at an age at which it is no longer a physiological food. There were 44 such boys, the details of their examination are as in Table VI.

TABLE VI.

Boys commencing raw milk before completing their eleventh year and now aged $13\frac{6}{12}$ years or over.

Number of boys	Average age at commencement	Average age now	State of second permanent molars
44	... $9\frac{11}{12}$ ...	$14\frac{5}{12}$ years ...	11 cavities in six boys (all very small cavities)

By comparison with Table I there should be 59 of these teeth carious, and by comparison with Table II, 77, though the latter comparison is not very accurate on account of the greater age at examination (by $1\frac{4}{12}$ years) of the Table II children. It would seem, therefore, that raw milk affords incomplete immunity when commenced at this age, possibly because it is not then a physiological food. At present one can only speculate as to whether it would afford complete protection for this tooth if given from birth, though in the one such case recorded it did so; as to how long the physiological period necessary for efficient immunity should be, is discussed later.

HYPOTHESES EXPLANATORY OF THE FINDINGS.

One is brought, then, to consider what the effect of raw milk on the structure of enamel may be and how it is caused. G. V. Black [10], after exhaustive inquiry, stated his conviction that "neither the density nor the percentage of lime salts, nor the strength, is in any degree a factor in predisposing the teeth to caries or inhibiting its progress." I have always thought that the important point is not the percentage of lime salts in the enamel so much as their stability, or the stability of the compounds which they form with the organic matrix; this is impressed on one because some

[1] Raw milk is supplied to this hospital.

animals, though they have relatively large channels in the "tubular" enamel of their teeth when newly erupted—and hence presumably a lower percentage of calcium content—yet seem to be as immune to caries as most other animals, even in a state of captivity. Dentine, too, with its much lower calcium content, becomes exposed by the process of attrition on the occlusal surfaces of the teeth of nearly everybody as they approach middle life, but it does not become carious.

Now, practically all children get a considerable milk ration during the first three or four years of life, but it is almost universally sterilized by boiling or pasteurized, both on account of the fear of infection with bovine tubercle and because it then keeps longer. These processes are believed to have no effect on the vitamin D content or salts present in milk, and to affect the protein to a small extent only; both boiling and pasteurizing, one knows, can destroy or alter the antiscorbutic principle, so that [11] "if very young children on an exclusively milk diet be given boiled milk alone, in a certain number of cases scurvy results." It is possible that other of the vitamin content may also be altered or destroyed by this means.

Boiling and pasteurization, then, by destroying or altering part of the vitamin content of milk, upset the vitamin balance and/or may in some other way so affect it as to bring about the deciding factor as to whether enamel shall prove resistant to caries or not. There is, too, the possibility that fresh raw milk may be a more vital[1] element than we are at present aware, as physiologically it passes direct from donor to recipient at body temperature, which is not a common procedure. The possibility also suggests itself that, dependent on their source, there may be different properties or strains of what are at present thought to be identical accessory food factors, and that some may have selective powers for building certain tissues. In any case, whenever a child is hand-fed, unless raw milk is given there is necessarily an unnatural balance of vitamins, and possibly other things, in its food over a prolonged period, coincident with the calcification of those teeth which later on show the greatest incidence of caries.

It is a well recognized fact that the teeth of primitive races show a smaller incidence of caries than those of civilized races, and that that smaller incidence is increased as civilization advances; it is also true that the women of many of these races suckled their children for from one and a half to three years, and in some cases considerably longer,[2] though it is believed by many that this was done partly with the idea of delaying the next pregnancy. With the coming of civilization this custom is apt gradually to fall into abeyance, though usually not till after their foods include those of civilization, some of which are known to be caries producing; it is probable that these factors together are responsible for the increased incidence of caries which invariably follows the advent of civilization. Perhaps Maoris and North American Indians are as good an example of this happening as one can find.

Concerning the former, Pickerill [12] records caries as being present in 2 only

1 "The newly born infant of a mother who has had measles previously is immune to attack, and this immunity . . . . lasts for two months absolutely and for four or five months relatively. If the child is breast-fed the duration of immunity, absolute and relative, is two months or so longer . . . . there is reason to suppose that small amounts of antibody pass in the maternal milk."—Gunn, William, "Practical Aspects of Measles," *The Practitioner*, vol. cxxviii, p. 22 (1932).

"It has been found by numerous workers that the number of bacteria in freshly drawn milk tends to decrease during the first few hours, when estimated by the total plate count. The apparent bactericidal power is active for a short time at 37° C., while at 15° C. the action is less marked, but more prolonged. The action appears to be specific for certain organisms in each sample of milk, but the milk from different cows affects different organisms. The apparent bactericidal power is destroyed by heat. A part of the reduction is due to agglutination of the bacteria, which is not detected by the method of plating out. Part also may be due to the antagonistic action of different species of organisms, and it is possible that part may be due to the leucocytes in the milk. It is also possible that certain bactericidal substances may be derived from the blood, and be secreted in the milk."—Vollum, R. L., "A System of Bacteriology in Relation to Medicine," vol. iii, p. 56 (1929).

2 Dr. A. H. Macdonald informs me that he has seen a native girl of six years of age in the act of being suckled in the Keeling (Cocos) Islands.

of 260 skulls of the uncivilized period, but that it is present in 95% children living under European conditions; Ploss and Bartells [13] state that they used to breast-feed their children for six years. North American Indians, whom John Mummery [14] showed to have had one of the lowest incidences of caries among primitive races, are stated by Ploss and Bartells to have breast-fed their children for twelve years. It is of interest to note that the use of milk as an article of diet was unknown to either of these races till the introduction of European civilization.

Consideration of the effects of raw milk on the teeth indicates that physiologically a longer period of breast-feeding than is usually practised in civilized communities is necessary for ideal dental development, and that raw cow's milk is an efficient substitute for the child's natural food.

Sir Frank Colyer [15] refers to "the phenomenal rapidity with which caries has increased in the last few decades"; it is a quadruple coincidence that in that period the hand-feeding of children, the treatment of milk by heat, the consumption of sugar[1] and refined starchy foods, and the incidence of dental caries have all reached their peaks of incidence among civilized peoples.

It is known that carbohydrates are the only foods which readily give rise to acid on fermentation in the mouth, and that the start of caries is by decalcification of the enamel; the presence of less stable enamel, brought about by hand feeding and the treatment of milk by heat, necessarily facilitates the action of such acid as is produced by the carbohydrate fermentation.

It would seem, therefore, that the coincidence of the first three of these peaks of incidence may well be the cause of the coincident peak in the incidence of caries. This is not meant to imply that no other factors are concerned in the incidence of dental caries. The possibility that raw milk may favourably alter the environment of the teeth by its effect on the oral secretions must also be kept in mind, though at present one has no knowledge that it does so.

Whatever the reasons, the teeth of the children of Groups I, II and III, and at B, provide an interesting study, it would seem from the dietary of some of them that an attempt is being made to produce caries and that it is unsuccessful.

It may be thought that in each group of children tabulated, a long enough period has not elapsed since the eruption of the teeth for the tests to be of sufficient value, but it is common knowledge that teeth are most likely to become carious very shortly after eruption.

Conclusions Concerning the Teeth.

The findings seem to show that if raw milk forms part of the diet from birth, so long as it is continued all enamel formed has some immunity to dental caries; that if it is not commenced till later, erupted but incompletely formed teeth may be similarly altered[2] though it may be to a lesser degree; it is problematical whether fully-formed teeth can be influenced, though one's original observations at B seem to imply that they may be. This seems to imply that an essential part of the immunity attained in this manner occurs in the period following the eruption of a tooth.

From the study of the boys of Table V it seems certain that if raw milk is not commenced till the third or fourth year, no protection is afforded to the deciduous teeth, though it is to the permanent teeth; so that at about the age of 8 or nine years both protected (permanent) and unprotected (deciduous) teeth may be present simultaneously. The converse may also occur if the conditions are reversed,

[1] At the beginning of the eighteenth century the average amount of sugar imported into this country was 3 lb. per head, at the beginning of the nineteenth century it had reached 22 lb. per head, in 1928-29 the consumption had reached 101·3 lb. per head; a considerable amount of the readily fermentable monosaccharide glucose is also consumed.

[2] The bulk of the enamel cannot of course be increased at this period, the finding applies only to its quality.

i.e., if raw milk be taken for only two and a half to three years after birth, the deciduous teeth are protected and but little, if any, protection afforded to the permanent teeth.

For ideal dental development in man it seems that raw milk should form part of the daily food till the age of 14 years is reached, as, by comparison, the clinical evidence in Table VI seems clearly to indicate that it has effect up to that age for teeth not yet completed. One has no exact knowledge as to how long or to what extent the immunity to caries thereby produced will last, but so far I have not seen a carious tooth in a person kept consistently on raw milk from before 12 months of age, though in none of the tabulated cases has there been any restriction as to sweets or other foods. Even so, breast-feeding is of such value in widening the developing arches of the jaws, that whenever possible a child should be so fed for at least nine months. At present it is uncertain whether this procedure protects the second permanent molar, but if milk affords protection to any of the teeth it seems reasonable for it to protect all; in the only tabulated case in which raw milk was given from birth and this tooth has erupted (now aged $19\frac{6}{12}$) it has done so; and in the cases tabulated in Table VI, when it was started at a later and a-physiological period, by comparison, it afforded partial protection only. If raw milk is not started till about the eighth or ninth year it seems to afford some protection to all the permanent teeth, though it is evidently not complete for the second permanent molar.

It would seem that the factors deciding whether a person shall be immune to caries are largely determined by the time the age of 7 or 8 years has been reached, because it is common clinical experience that if a person is caries-free up to that age he often remains relatively free for a prolonged period. The three caries-free children of Tables I and II are confirmatory of this.

But one must realize that freedom from caries in early life may be caused in two ways: artificially, by the parental restriction or control of the refined carbohydrate intake, and physiologically, by the natural immunity attained in the manner I have described. When it is due to the former, the acquisition of pocket money and the proximity of the tuck shop very often terminate the freedom at about the twelfth or thirteenth year, but immunes do not become carious at this age.

Freedom from caries and immunity to it are, of course, very different things, though both may be present simultaneously in varying degree.

There seem also to be physical differences of a protective nature in teeth developed under these circumstances; deep and sharp fissures and pits appear to be absent from the occlusal surfaces and other aspects where they occasionally occur, and the cusps are less sharp and pointed; these appearances are best seen in the first permanent molars and, at their maximum, make them almost typically bunodont teeth, possibly these changed appearances are brought about by increased bulk in the enamel.

It would not be surprising if interglobular spaces were entirely absent from the dentine of such teeth, even when they are hypoplasic, though ordinarily they are practically constant in young civilized teeth.

Considerations Bearing on the Thesis.

It has been shown [16] that of 9,082 English children examined between the ages of 5 and 6 years, and of whom rather more than one-third were breast-fed, the breast-fed child had a slightly lower incidence of caries; the difference, however, was very small. There is no record as to how long breast-feeding was maintained or, what is perhaps of more importance, what the nature of the food was subsequent to its cessation.

The relative freedom of animals from caries and, more particularly, the difficulty of producing caries in them, may be partly due to their being suckled for the

physiological period requisite for the efficient formation of their dental structures, but there is no doubt that the physical nature of their food is also their protection.

Wild animals are wholly or partly dependent on maternal milk for their sustenance for about a quarter of their growth period. Domesticated carnivores, such as dogs, are weaned and then hand-fed from an earlier date, about two months, this being reckoned by veterinary surgeons as one-sixth of the growth period, but in the wild state the young are unable to obtain sufficient suitable food to sustain themselves independently at that age and so are partly dependent on maternal feeding for a longer period. So also with ungulates; indeed in the economics of animal husbandry, if it is desired to produce particularly fine foals or calves, as for race horses or show purposes, the period of maternal feeding is allowed to continue for ten months, or rather more than a quarter of the growth period of these animals, which is taken as three years. Commercially the period of maternal feeding of domesticated ungulates is an elastic one, its duration depending on the purpose for which the animal or its progeny is required. By comparison, the period of maternal or raw milk feeding customary with civilized peoples seems much curtailed. By comparison too with other mammals, the growth period of man is excessively long as compared with his ultimate longevity, taking this latter as the Psalmist's three score and ten years.

The actions and thoughts of other nations to whom the use of cow's milk was unknown, are of interest on this subject of maternal feeding.

The Eskimos, among whom the incidence of caries was one of the lowest known among the human race till they came into contact with civilization (only one in sixty-nine of their skulls showed any incidence of caries [14]) according to Nansen [17], who lived there in 1888, often breast-fed their children until 3 or 4 years old and, he continues, " I have even heard of cases in which children of 10 or 12 continued to take the breast. A European at Godthaab told me that he had seen a dashing youth of 12 or so come home in his kaiak with his booty, rush up to his home, and there consume a biscuit, standing between his mother's knees, and drinking, from time to time, from her breast." Ploss and Bartells [13] state that the Eskimos of Smith Sound breast-feed for seven years, and those of King William Land for 14 or 15 years.

In uncivilized Greenland, if a child's mother dies and a nurse is not available, it is killed, as a rule by exposure or throwing it into the sea, as there is no alternative means of nourishment for it.[1] There have been centres of civilization in Greenland, certainly since 1728; till then, refined carbohydrate, either as starch or sugar, seems to have been entirely absent from their dietary; but, being a scattered and nomadic race, and occupying (to us) an inhospitable terrain, civilization has spread relatively slowly, especially along the eastern coast.

The Japanese, among whom, till sixty years ago [18], the use of milk as an article of diet was unknown (though there are references that they did so use it for a period terminating at the beginning of the fourteenth century) have a proverb: " one sho (1·8 litres) mother's milk is worth 3,770 measures of linen, 23,000 bundles of rice straw and more than 10,850 bushels of rice." I am unable at present to obtain information of their incidence of caries either as children or adults prior to the introduction of Western civilization; according to Ploss and Bartells [13], they breast-fed their children for three to five and ten years, and Ashmead [19] states that " the children are suckled until the sixth year," so if the inferences drawn from the clinical facts presented are correct, their incidence should not, by virtue of their proverb and practice, be a high one. Their diet, of course, is very different from that of the Eskimos. Rickets also is said to be practically non-existent among them.

[1] Dr. H. C. Gilmore informs me that under similar circumstances and for the same reasons native children die in the Belgian Congo, where the use of milk as an article of food is unknown.

African races, generally speaking, breast-feed their children for from two and a half to three years, so, if this hypothesis is correct should have the deciduous teeth immune to caries, but not the permanent teeth ; I am informed by H. Stobie, who has experience with such Kaffir and Zulu children, that he has never seen caries in their deciduous teeth, but only in their permanent teeth, and notably in the first permanent molars. The incidence of caries in the permanent teeth would seem therefore to be entirely dependent on the physical nature of their foods and the presence or absence of refined fermentable carbohydrate in their dietary.

It is possible that the time that elapses between milking and the delivery of milk to the consumer may allow changes to take place, produced possibly by bacterial growth or effect, or by other means, such as may lessen its nutritive or growth efficiency ; one has no knowledge that it does so, but as already mentioned, physiologically, milk passes direct from donor to recipient at body temperature.

The foregoing does not at all alter one's conception of the causes of dental caries, but urges the probability that the composition and form of some human enamels make them more resistant to the onset of caries than others, and statistically shows evidence that this increased resistance can be caused in the manner stated.

It is usual to consider that all calcified tissues are influenced in a similar manner by the same food substances, but there are factors which indicate that this is not entirely so : for instance, the major portion of enamel calcification occurs during the first twelve years of life, and intensively at that period when raw milk is a physiological food, whereas bones increase in size and density mainly when the individual exercises choice in the foods eaten, and milk is no longer a physiological food. This would seem to imply that the vitamin balance and other constituents of raw milk have a special significance with reference to enamel and tooth calcification, but that bones should react rather to such accessory food factors as are obtained from foods selected by the individual and by extraneous means such as exposure to ultra-violet rays. The words of F. William Fox concerning rickets, already quoted, have a confirmatory bearing on this.

It is a fact that the nourishment of young by milk is almost entirely confined to those vertebrates (chiefly diphyodont) in which one set of teeth has to last most of the animal's life ; vertebrates having polyphyodont dentitions, in which each tooth is functional for a relatively brief period only, find their food independently from birth or hatching. There are, however, exceptions, as a few mammals are functionally or entirely edentulous, though these are mostly looked on as being evolutionarily retrograde ; and among reptiles one Order[1] (which however only comprises one species) and one Sub-order[2] are monophyodont.

Among living vertebrates, teeth with " closed " apices (as in man) or of continuous growth are entirely confined to mammals, though some teeth of the piscine Family Sphyræna certainly have a tendency towards closed apices, their dentine structure[3] is however, with one exception, unlike that of any in the whole mammalian class.

Of interest also are the conditions in such primitive mammals as the Monotremata, the few varieties of which possess no nipples or teats, the milk glands opening by a number of small pores in a cup-like depression in the skin of the abdomen, from which, presumably, the young lick the secretion ; coincident with these rudimentary mammalian attributes, one Family of these animals is edentulous and the other possesses teeth only when young ; members of this latter triturate their food with horny plates, which are developed immediately beneath the sites once occupied by the teeth, throughout the greater portion of their lives.

1 Rhynchocephalia.

2 Rhiptoglossa.

3 Different textbooks state that the dentine of Sphyræna is osteodentine and vasodentine respectively ; an examination of it from sections made for the purpose of comparison in this paper clearly demonstrates that it is neither, but is a complex and very beautiful form of plicidentine, not unlike that found in the rostral spines of the Elasmobranch fish Pristis.

Concerning Lymphoid Tissue.

The effect of raw milk on the teeth led to inquiry concerning the incidence of such other abnormalities of childhood as adenoids and enlarged tonsils in these children, and the incidence of these diseases is of interest and, it may be, of importance. In the 40 children (Table III) who commenced raw milk at the average age of $4\frac{1}{2}$ months, two only (5 per cent.) had required the tonsil and adenoid operation; of the 33 children (Table IV) who commenced raw milk at the average age of $1\frac{9}{12}$ years, 10 (30 per cent.) had had the operation performed. This lesser incidence in those commencing raw milk at the earlier age, is of interest when one considers the large percentage among all of these children who were subnormal on admission; one has no information of the manner of feeding of any of them prior to this date or whether they had any breast feeding at all; but in view of the nature of their subnormalities, which were in some cases serious, it is almost certain that their feeding prior to admission was bad.

On this account it is impossible to estimate what the incidence of need for this operation might have been if raw milk had not come into their diet scale, as one knows of no groups of similar children with which to compare them, but the ordinary incidence of the operation at A, among all children admitted, from ages 2 to 6 years, over three consecutive years was approximately 24 % (133 operations in 564 children). The possibility suggests itself that the incidence of need for this operation might be reduced to a minimum in children kept from birth on this physiological food and/or its efficient substitute, as, from its early age incidence, it is obviously caused by factors acting during this earliest period of life. It even suggests that there may be some relation between the incidences of enlarged tonsils and carious teeth, and that, for instance, a child brought up on raw milk should have neither; if it were not commenced till 12 to 18 months old, it may get enlarged tonsils but should have good teeth; and if not having it till 3 or 4 years old, or at all, it would probably have both, though at the former age caries should be confined to the deciduous teeth; the figures quoted, however, are far too small to hypothesize on to such extent with any degree of accuracy.

Conclusion

The facts recorded concern the effects of raw milk. I am not aware of any comparable body of children who have been similarly brought up on sterilized or pasteurized milk, and so have no exact knowledge whether effects comparable with those recorded might be brought about by such means, but such evidence as is available and inference, indicate rather that they would not be. Unless due care is taken there are risks in taking raw milk, and as things are at present, only such milk as "Certified" or "Grade A (Tuberculin Tested)"[1] should be so used.

Different authorities give the incidence of tuberculous disease in milch cows [20] as 40 %, adding that the real proportion is probably considerably higher; and in adult stalled animals [21] as from about 30 to 70 or 80 or even to 100 %. Comparatively few of these animals, however, yield tuberculous milk, but as the disease is progressive they are all potential yielders of it should they live long enough. Not more than about 2 % of all milch cows in this country are believed to yield tuberculous milk.

It is practically certain that the great majority of human infections with bovine tubercle are conveyed by means of cow's milk, usually during childhood; such infections, however, very rarely, if ever, produce pulmonary tuberculosis, and do not endanger life to anything like the same extent as do infections with the human type of organism, against which they are regarded as being protective.

Many people are under the impression that, because they have provided "Certified" or "Grade A (T.T.)" milk for the purpose, their children have been

[1] The use of these special designations was provided for under The Milk and Dairies (Amendment) Act, 1922, and the Milk (Special Designations) Order, 1923.

brought up on raw milk, whereas the facts are frequently otherwise. In the preparation of infant foods when the child is weaned, the instructions for preparation are usually, if not always, that the milk (or water in the case of a milk food) should be added when boiling; this, of course, defeats the real object aimed at in the provision of high grade raw milks, and such children would, more likely than not, have carious teeth.

Of a total of ten proprietary infant foods in use in the children's wards of a large London Hospital, instructions on nine of them are that they should be made up with boiling water or boiling milk, the tenth was a dried milk food to be made up with boiled (but cooled) water. "Certified" and "Grade A" milks when used in these wards are also boiled for ten minutes.

It is extremely difficult to get a really accurate history as to a child's early milk feeding in any private home, and the most reliable private home test is when children have had raw milk to drink cold daily since they were four years old, and the effects on the permanent teeth are noted, though the maximum effects on the first permanent molars, with regard to shape, will not be seen even then.

Obviously, physiological principles must be one's guide, and these indicate that milk should at no time be raised above body temperature. Obviously also, the child's diet should otherwise be well balanced, and fruits, also raw, included at an early age. A comparison with Tables I and II will show definitely however, that raw milk is the essential factor, as otherwise the diets of all children tabulated over five years old have been very similar.

I have no knowledge what importance attaches to the freshness of the raw milk taken, but about 90% of the children quoted use it within twenty-four hours of its being obtained from the cow.

One fact which emerges conclusively from this investigation is that well-formed teeth, having apparently a considerable degree of immunity to dental caries, though this immunity is at present of unknown limits, may be developed entirely independently of ante-natal care; though ante-natal care, in addition, should make them even better.

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Discussion.—Mr. E. B. DOWSETT (President) said that he had always been interested in the subject of breast feeding and the incidence of caries, and his personal observations confirmed those of Mr. Sprawson. He had pointed out that the teeth of children fed on cod-liver oil and pasteurized milk and exposed to sunshine were more carious than those shown to have had raw milk. In a recently published work[1] the teeth of children who had suffered from rickets were compared with those of a control group, of similar age and social class, who had not, and it was found that hypoplasia of the permanent teeth was almost confined to the rachitic group, in spite of the fact that the amount of caries in the permanent teeth appeared only slightly greater in the rachitic group than among the controls, and the difference was such that it could not be held to indicate that vitamin D deficiency in early childhood was an important factor in determining the development of caries. The amount of caries of the temporary teeth was about the same in the rachitic and control groups. In the *Journal of the American Dental Association*, investigations carried out by what was termed the Michigan group of scientists, were reported on much the same lines as those of Mr. Sprawson. Children in orphanages, fed on raw milk, green vegetables and fresh fruit, had shown infinitely less caries than those not so fed, but no claim had been made that it was raw milk alone that had produced the results they had attained.

Mr. J. LEWIN PAYNE said that Mr. Sprawson had stressed the importance of the quality of the milk as well as that of its freshness. The coincidence of rickets and defective structure of the teeth was well known, and the work of Miss Ethel Luce, at the Lister Institute, might be quoted in this connection. It had been found that when a cow was fed on dry fodder and kept in a dark stall for six months the growth-promoting and antirachitic properties of the milk were poor; when the cow was fed on fresh green fodder in a dark stall for two months the growth-promoting effect was good but the antirachitic effect was poor. On dry fodder, out of doors in the sun, the growth-promoting properties were poor and the antirachitic moderate.

On the other hand, when the cow was out on pasture after three months both were good, and it was estimated that the "good" milk had about four times the activity of the "poor." It would appear that the growth-promoting factor in milk depended mainly on the diet, whilst the antirachitic factor was determined partly by the diet and partly by the amount of light to which the cow is exposed. These factors might have an important bearing upon the building up of a normal structure in teeth.

The quality of the milk might well influence immunity to dental disease not only as an important factor in building up the structure of teeth but also because through its agency the secretions of the mouth might be maintained in normal activity and thus the environment of the teeth be kept healthy.

Normal nutrition, normal occlusion and normal mouth hygiene were the important factors in the prevention of dental disease, and to some extent, at all events, these factors were inter-related.

Mr. A. T. PITTS said that he had gathered that raw milk, as opposed to pasteurized milk, conferred an immunity or freedom from dental caries. He believed that pasteurized milk had been held not to affect the vitamin D content. A point of some significance was that the children referred to by Mr. Sprawson lived under institutional conditions, involving a diet routine not found in the average household. It would strengthen his conclusions considerably if the same conditions were found to apply to children living in private homes. Experience had shown that the period of suckling in the hospital class was longer than in the middle or upper classes.

Mr. GEORGE NORTHCROFT said that about twenty years ago "Certified" milk was brought into use under the ægis of the National Clean Milk Society. All three of his own children were breast-fed for nine months, and thereafter had "Certified" milk—but all had caries. He did not think Mr. Sprawson had quite proved his point that raw milk was necessarily an efficient antidote to caries.

[1] "Vitamin D deficiency, dental caries, and tonsillar enlargement—a clinical investigation of some late effects of rickets," by Helen M. M. Mackay, M.D., M.R.C.P.Lond., Physician to the Queen's Hospital for Children, London. With the collaboration of S. F. Rose, M.R.C.S., L.D.S.Eng., Dental Surgeon to the Hospital, *Lancet*, (ii) 1230, 1931.

Mr. SPRAWSON (in reply) said that, omitting three exceptional cases, having found a coincidence which worked out 100 per cent., not only for one, but for *all* teeth erupted subsequent to raw milk being started before the age of 6 years, he had considered it a matter which should be brought to the notice of the Section. So far, knowledge of the vitamins and their nature was very limited. He agreed with Mr. Lewin Payne that there was little doubt that the vitamin quality of milk varied considerably according to the feeding of the cow—whether it was stall-fed or pasture-fed. The main groups of the children concerned in the investigation lived in the country, and it was presumed that the quality of the milk provided for them was satisfactory. In reply to Mr. Pitts, vitamin D was believed to be unchanged either by pasteurization or by boiling. Vitamin C, it was known, was destroyed by boiling and was altered by pasteurization. He did not imagine that the ideal calcification of teeth depended entirely upon one vitamin. As to breast feeding of children in this country, it was probable that the average poor mother who suckled her child could often be compared with the stall-fed animal referred to by Mr. Lewin Payne. Mr. Northcroft's remarks were difficult to assess for comparison with the cases quoted, because "Certified" milk was often not used in the raw state in the household, and it was of raw milk that he had recorded observations; so far as he knew, Government "Certified" milk, as understood now, had only been in existence since 1923. Up to the present he had not met any carious teeth in children who had been brought up on raw milk. As Mr. Lewin Payne had stated, the quality of "Certified" milk might vary very greatly, and so constitute an important factor in the results obtained.

Section of Dermatology.

[*December* 17, 1931.]

Cutis Verticis Gyrata.—F. PARKES WEBER, M.D.

D.S., aged 31 years, of a Polish Jewish family. This is the original case shown by Dr. M. G. Hannay (*Brit. Journ. Derm. and Syph.*, 1923, xxxv, p. 451), and by Dr. A. C. Roxburgh (*Proc. Roy. Soc. Med.*, Section of Dermatology, 1926, xix, p. 14), and by Dr. A. M. H. Gray (Annual Meeting of the Brit. Assn. Derm. and Syph., July 31, 1930). The patient has likewise been under the care of other physicians, notably for his generalized pruriginous dermatosis. This dates from childhood, though the accompanying lichenification and general thickening of the skin were doubtless not present at first. The actual cutis verticis gyrata, so well described and illustrated by Dr. Hannay, seems gradually to have developed about ten years ago. I would draw attention (as has been previously done) to the gyrate folds of the forehead, as well as of the hairy scalp.

The obvious spinal scoliosis seems to have been first observed about fourteen years ago. There is a slight blood-eosinophilia and the monocyte count is relatively high.

The Wassermann reaction is negative.

Brachial blood-pressure, 145/115 mm. Hg. The urine contains a trace of albumin and a few granular and hyaline tube-casts.

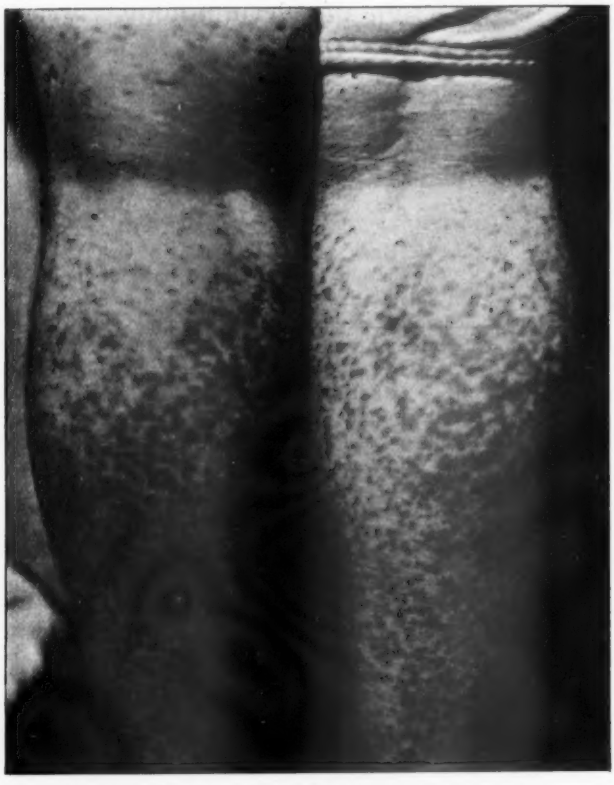
It should be specially mentioned that, though the patient did not go to school (owing to his skin trouble), and received very little education, his general intelligence seems to be quite up to the average. He is certainly not acromegalic; by radiographic examination his pituitary fossa is found to measure 10 by 8 mm., and has a normal outline.

Urticaria Pigmentosa (telangiectatic variety) in an Adult.—F. PARKES WEBER, M.D.

The patient, aged 30 years, is a fat woman of Russian-Hebrew parentage, with nothing special in her past or family medical history, excepting the skin condition in question. This is the best seen on the calves of the legs and on the flexor surfaces of the forearm. The elementary lesions are brownish-red, slightly raised macules, from 2 to 3 mm. in diameter, discrete on the forearms, but tending to be confluent on the legs (figure). Slight brown pigmentation is left when the lesions are rendered anæmic by pressure under glass. The spots appeared rather suddenly on the legs about five years ago (about the time when she was becoming fat), and simultaneously, but more gradually, on the arms. No "urticaria factitia" can be excited. The patient has not been troubled by itching and has never had an attack of ordinary urticaria. The blood-count is normal, excepting that the lymphocytes constitute 45 per cent. of the total white cells; no excess of mast cells. Otherwise nothing abnormal is found by general examination, including blood-pressure and urine. The

blood-serum gives negative Wassermann and Meinicke reactions. No fresh spots have appeared for a long while, and it is possible that the condition will gradually become less obvious (but very little is known as to ultimate subsequent history in cases of urticaria pigmentosa in adults).

The eruption in cases of this type is closely allied to, if not identical with, the disseminated erythemato-telangiectatic plaques[1] which have been classed under



Urticaria pigmentosa (telangiectatic variety) in an adult.

"Télangiectasies essentielles en plaques acquises" by L. Brocq (*Précis-Atlas de Pratique Dermatologique*, Paris, 1921, p. 1043) and others in France. I regard Osler's "telangiectasis circumscripta universalis" as of the same nature (cf. F. P. Weber, *International Clinics*, 1931, series 41, ii, p. 131), though definite factitious urticaria could be excited in his patient; and what I have termed "telangiectasia macularis eruptiva perstans" (Weber and Hellenschmied, *Brit. Journ. Derm. and Syph.*, 1930, xlii, p. 374) is doubtless also of the same nature.

1 I here employ the word "telangiectatic" in its exact sense, as indicating that in the lesions there is *permanent* dilatation of the *terminal* peripheral blood-vessels (i.e., the capillaries), just as one speaks of the coloration of a "port-wine nævus" being due to a telangiectasis of its capillary network, though the individual dilated blood-vessels cannot be distinguished by the naked eye.

Scleroderma in a Tuberculous Subject.—A. M. H. GRAY, C.B.E., M.D. (President).

The patient, a hospital nurse, aged 30, was operated on in 1925 for some disease of the ankle. In the following year she had pleurisy, pneumonia and hæmoptysis; tubercle bacilli were found in the sputum. She was sent to Brompton Hospital and was treated for three years with artificial pneumothorax. Later she had phrenic evulsion performed on the left side. While in Brompton Hospital she had acute rheumatism and tonsillitis; the tonsils were removed later.

During 1929 she was given weekly injections of sanocrysin, consisting of 0·1 grm. for a year. Since then her condition has been quiescent and she has not required any further treatment for the tuberculosis.

In January, 1931, she had an attack of boils in the axillæ, and styes on the eyelids. Following this a rash began to develop, spreading from the armpits across the upper part of the chest. Later, a similar rash appeared on the hips and on the thighs.

Present condition.—There is a symmetrical eruption confined mainly to the region of the shoulder and pelvic girdles. The rash extends over the top of each shoulder like a cap and round into each axilla, extending down the upper two-thirds of the arms on the front and inner aspect; on the right side this extends down as far as the elbow. There are a few faint patches on the right forearm. Also on the right side are a few scattered patches running down the chest wall.

In the front, the eruption over the two shoulders joins over the sternum and lower part of the neck. The skin over the shoulders and arms is a brownish-red colour and is scaly. The pigmentation does not entirely disappear on pressure. At the margin of the patch small, slightly scaly, pinker areas may be seen. The general patches are sharply defined. The skin over the clavicles and centre of the manubrium is shiny and dead white in colour, resembling a scleroderma, and in many places shows a number of scattered white dots. Similar patches are developing on the patches on the arms and in the bend of the right elbow.

A similar brownish-red, slightly scaly eruption is present over the outer side of the hips and thighs, running forward across the front of the thighs towards the inner aspect. Central atrophy is also present in this region. There are also similar patches behind both knees.

The patient has had four doses of X-rays, of one third of the skin unit, at weekly intervals, over the patches on the thighs. Scaling has disappeared in the areas treated, but the pigmentation remains. The patient is now having X-ray treatment for the shoulder patches.

A section from the erythematous area on the outer side of the left thigh shows a marked lymphocytic infiltration along the vessels throughout the dermis and some islets of these cells in the fatty lobules of the hypoderm. The endothelium of the smaller vessels is proliferated and some vessels are thrombosed. The papillary layer is swollen and there is a tendency to flattening out of the papillæ. There is some patchy œdema of the basal layers of the epidermis with slight inter- and intracellular œdema; this is associated with a patchy parakeratosis.

Discussion.—Dr. H. MACCORMAC said this case showed that it was impossible to determine whether "white spot" disease arose from lichen planus or scleroderma. The white spots in the present instance were identical with the "white spots" in a case exhibited at the last meeting; and yet one was definitely an example of scleroderma and the other of lichen planus, as proved by the characteristic eruption elsewhere.

Dr. E. STOLKIND said that he had a case of scleroderma which had lasted nineteen years. Tuberculosis of the spine had developed six years ago. The scleroderma had not progressed

very much in the nineteen years. The patient had, in addition, Raynaud's disease, telangiectases, and paralysis agitans.

The PRESIDENT, in reply, said he thought the case would be interesting to members because of that shown by Dr. Goldsmith some time ago, with a definite tuberculous appearance, histologically in a pre-sclerodermatous lesion on the leg. Reference was now being made in textbooks to the morphœic tuberculide. He did not know whether this lesion would be regarded as of that type.

Sporotrichosis.—A. M. H. GRAY, C.B.E., M.D., and G. W. BAMBER.

Patient, female, aged 43. On or about September 13, 1931, she was bitten by a boa-constrictor on the left wrist. This animal has since died from what is described as "canker" of the mouth. On October 9 a spot appeared on the site of the bite and this gradually increased in size, eventually beginning to ulcerate in the centre. At the beginning of November several raised, red lumps appeared on the front of the left forearm.

The patient was first seen by us on November 30. She had a raised, red, oval patch, about 1½ in. by ¾ in. on the radial side of the left wrist; it was softly infiltrated and ulcerated in the centre. At the edge a number of raised, red lines and small

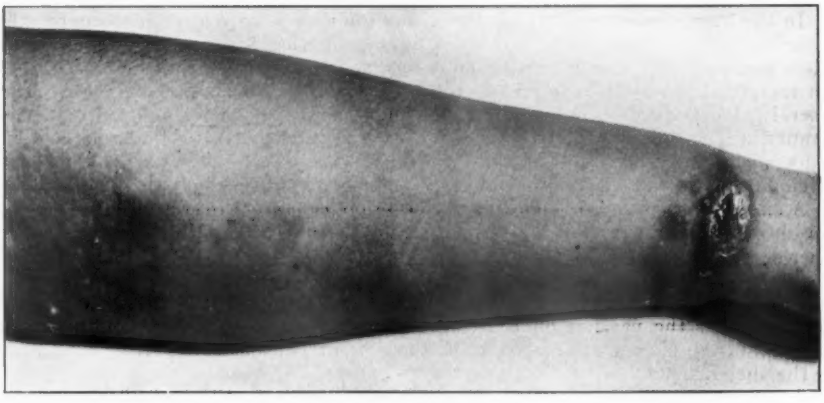


FIG. 1.—Sporotrichosis.

papules were seen radiating outwards. On the front of the forearm a number of subcutaneous nodules, from the size of a pea to a sloe, could be felt, and two larger ones were visible as a red swelling protruding from the surface. The epitrochlear and axillary glands were enlarged.

The patient stated that she had a hæmoptysis in October and the chest was therefore examined, but no abnormal physical signs were detected, nor was anything abnormal discovered on X-ray examination. The blood-Wassermann reaction was negative. There have been some flat lichen planus patches on the ankles since 1921.

Cultures on glucose agar and on Sabouraud's medium were incubated at 25° C. Growth was apparent in two days' time as small white points which soon developed radial striations. On Sabouraud's medium the growth became raised and convoluted,

the colour rapidly turning buff, then dark brown with a black centre. On glucose agar the growth was less abundant and showed practically no change in colour. On glucose broth the growth formed a thick white skin on the surface. This skin was made of thin, branched, irregularly septate mycelium bearing lateral conidia.

It is extraordinary how few such cases have been shown in this country previously, there have been only five shown here; four of these were exhibited by Dr. Adamson; and Sir Ernest Graham-Little showed the other. Two have been published in Scotland, one of them by Sir Norman Walker, while Dr. Beatty, of Dublin, published an Irish case of the condition in the *Journal*. It is a very rare disease in this country.

Much has been written recently as to the ways in which this infection is transmitted. Most of the recent American papers indicate that it takes place in market gardeners owing to their receiving scratches from trees, especially the barberry bush, but some cases occur from animals.

Discussion.—Dr. H. G. ADAMSON said that in order to obtain cultures of sporothrix it was advisable to withdraw from an unbroken gumma 0·5 c.c. or more of pus, to squirt it over the surface of a sloped glucose-peptone-agar tube, and to keep the tube at room temperature and uncapped. In from six to ten days white points (which soon became black or dark brown) appeared here and there on the spread-out pus. From these subcultures were made. It was difficult to obtain cultures by merely using a platinum loop, and possibly for this reason the diagnosis of these cases was sometimes missed.

The PRESIDENT (in reply) said that the histological section showed that, in addition to dermic infiltration—with its characteristic giant cell grouping—there were a number of intradermic and subdermic abscesses, and this was interesting in view of the different types of lesions met with.

Sir ERNEST GRAHAM-LITTLE, M.D., showed the following cases :—

(1) **Parapsoriasis.**—Patient, a man aged 47, was born in South Africa and brought to England at the age of 6. At present nearly the entire surface of the body is involved in a superficial exfoliation, but without any induration or ulceration. The condition has persisted for thirty years. It seems to have been rendered worse by the application of ultra-violet light about 12 or 18 months ago. There is general enlargement of glands but no other indication of illness.

Dr. H. W. BARBER said that he, the speaker, had not been responsible for the giving of the ultra-violet light; he had given injections of gold and novarsenobillon.

(2) **Poikiloderma of Civatte.**—Patient, a woman aged about 40, a native of South America and lived there until two months ago, when she came to England on a visit. Her skin is naturally a dark olive colour, obviously with a touch of black blood. The deeper pigmentation concentrated chiefly over the malar eminences began to be noticeable after her arrival in England. There is no pigmentation elsewhere and her general health is apparently excellent.

(3) **Cicatricial alopecia** in a girl aged 6. Three years ago this child suffered from a severe inflammation of the scalp, diagnosed at the time as cellulitis, but without any evidence of operative treatment, and it would seem to have been rather a surface impetigo. There has been complete loss of hair over large areas of the scalp which now show definite cicatricial atrophy, but there is no trace of any suppuration at the present moment.

Discussion.—Dr. H. MACCORMAC thought the condition was the result of favus. In old-standing cases in adults one could still find hairs containing the mycelium, although the disease had become quiescent.

Dr. G. B. DOWLING said he considered that the alopecia was the result of severe impetiginous dermatitis. He had observed a similar case in a young woman, aged about 25, who had been under his care for some years and who suffered from chronic blepharitis

and impetiginous eczema of the ears. In childhood she had apparently suffered for years from chronic impetiginous eczema of the scalp; this had eventually cleared up, leaving a large area of cicatricial alopecia, which now bore a striking resemblance to folliculitis decalvans.

In the present case there was a history of severe impetigo of the scalp which had preceded the alopecia and had occupied the same area. He could not agree with the diagnosis of favus, for that condition would hardly have cleared up in a relatively short time without treatment.

Seborrhœic Verrucæ and Multiple Basal-celled Epitheliomata.—KNOWSLEY SIBLEY, M.D.

T.S., a man aged 65, has a number of so-called seborrhœic warts scattered over the trunk, both back and front. On the right side of the chest there is a typical superficial, non-ulcerating basal-celled epithelioma, about the size of a penny, which the patient says has been coming for about ten years. There is a large superficial ulcer over the right scapula, which he says occurred on a mole which was present at birth. He says that he had a red spot on the left side of the nose, also present from birth, which had been growing for the last ten years, and some six weeks ago, as it was increasing rather rapidly, he had it removed at the Cancer Hospital, and it proved to be rodent ulcer.

The biopsy of the large lesion over the scapula showed the typical histological structure of a superficial benign erythematoid epithelioma (Little).

Some of the warts over the shoulders, and one in particular, are showing marked changes to the naked eye and signs of becoming epitheliomatous.

Histological reports (Dr. I. K. MUENDE).—Section 1.—Verruca senilis: The epidermis shows acanthosis and hyperkeratosis, with the typical intradermal cystic spaces containing horny masses.

Section 2.—Superficial benign erythematoid epithelioma. The section shows the "rodent" cells taking origin from a rete peg.

Discussion.—Dr. H. MACCORMAC said he was not convinced that the malignant lesions originated in the seborrhœic warts. It was outside his experience to observe a generative change of this kind in a genuine seborrhœic wart. One "wart" looked red, and it would be interesting to see the microscopic appearances. Elsewhere he observed a rodent ulcer and a "wart" side by side and, in another position, a rodent ulcer growing on skin free from any warty changes. In making the above statement it would be understood that he drew a distinction between seborrhœic warts and keratosis senilis, as the latter was a genuine pre-malignant lesion.

Dr. H. G. ADAMSON said that he did not think it probable or even possible that multiple rodent ulcers could arise from so-called "sebaceous warts." Both were present in this case, but he thought they had no connection. Sebaceous (or senile) warts were actually neither sebaceous growths nor true warts. Although they were exceedingly common, he believed that they had never been known to become malignant, unlike the keratosis senilis of the face and hands with which they were sometimes confused.

Paget's Disease of the Nipple Associated with Diffuse Carcinomatosis of the Chest Wall.—H. W. BARBER, M.B.

C. J., female, aged 47. The patient's present condition is too serious to justify my bringing her to the meeting, but I am showing photographs, which give a fair idea of the clinical appearances, and also a section taken from the skin of the chest near the left axilla. I show photographs of a similar case which I saw some years ago also in a middle-aged woman; in this case, however, there was not Paget's disease of the nipple.

My present patient first noticed a "crack" in the left nipple about a year ago; it apparently healed without treatment. Six months ago she again noticed a "crack"

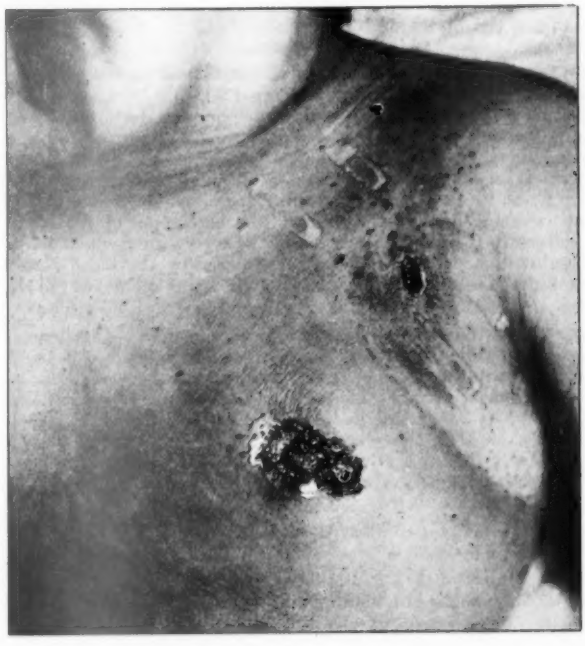
in the same nipple, which gradually developed into the condition now seen. She cannot date the onset of the diffuse carcinomatosis that now involves the skin of the chest.

When first seen a few weeks ago there was observed :

(1) Characteristic Paget's disease of the left nipple.

(2) A stony hardness of both breasts. It is difficult to say to what extent the breasts themselves are involved, owing to the induration of the skin over them.

(3) A diffuse thickening of the skin of the chest wall more marked on the left than on the right, extending downwards to the upper abdomen, upwards to the base



Paget's disease of the nipple associated with diffuse carcinomatosis of the chest wall.

of the neck, to the left axilla, and backwards over the dorsal aspect of the chest. There was some hard œdema of the skin of the left arm, doubtless due to obstruction of the lymphatics in the axilla.

(4) The bluish-red discoloration of the affected skin recalling that seen in erysipelas (carcinoma erysipelatodes of Rasch).

(5) Scattered here and there, but most numerous on the left side of the chest below the axilla, several small, hard, slightly raised nodules, clearly, I think, formed by carcinomatous deposits in the lymphatics. (These were more evident in my other case.)

The patient was admitted to hospital, and was found to have a large pleural

effusion, which has been tapped. As operative and radio-therapeutic treatments were obviously out of the question, she has been having injections of posterior pituitary extract and theelin. At the present time the discoloration of the skin is less, and there is some diminution in the infiltration, but of course I regard the prognosis as hopeless.

Clinically these two cases correspond to that published by Rasch (*Brit. Journ. Derm. and Syph.*, 1931, xliii, 351) under the title of "Carcinoma erysipelatodes." In his case, as in my first one, there was no Paget's disease of the nipple.

Report on section.—The biopsy was made from the skin below the left axilla and included some small raised lymphatic nodules. Groups of carcinoma cells can be seen lying in dilated lymphatics and blood-vessels. One of these is seen just beneath the epidermis, which is bulged outwards and thinned. This doubtless corresponds to one of the above nodules.

Dr. W. N. GOLDSMITH said he was much interested in Dr. Barber's cases, in view of a case he himself had shown before the Section on December 20, 1928,[1] that of an old lady who had a sudden inflammation on the top of her scalp; she thought it started from an insect bite. It was brilliant red, and spread over the scalp, causing the hair to fall out. He did not diagnose it as carcinoma for a long time, but a section of it showed very large spheroidal cells throughout the skin, characteristic of breast carcinoma. Only then did he suspect the breast, which was found to have been removed. There was carcinoma of the cuirasse type about the scar and running up into the axilla. The carcinoma of the top of the head was certainly erysipelatoid, in the sense that it started as a very intense and acute inflammation, with erythema, and it must have been conveyed thither by the blood-vessels; it could not have reached the top of the scalp by way of lymphatics, and moreover the lymphatics of the neck were normal.

The PRESIDENT said that recently he had a patient sent to his department who had had her breast removed not long previously, and she was beginning to develop some redness round the scar. On careful inspection it could be seen to be composed of very fine lines travelling outwards from the scar. The section showed the same sort of picture as Dr. Barber's section did. The bulk of the malignant cells were lying in the lymphatics. He did not know whether the blood-vessels were involved. It was certain, however, from present knowledge of the disease, that both structures must be involved sooner or later. The primary spread was through the lymphatics, then, secondarily, through the blood-vessels.

Leiomyoma.—W. N. GOLDSMITH, M.D.

I. P., female, aged 26.

Present condition.—The arms and forearms (mainly the extensor aspect), the thighs and legs (both aspects but leaving out the flexures of knees) and the shoulders and sternoclavicular region present a profuse eruption of small, round or oval, hard elevations, some white, some deep red, a few discrete but most coalescing into extensive sheets. The overlying epidermis is normal. The individual nodules have mostly a long diameter of about 5 mm. The left cheek is thrown into thick raised folds of rather fawn colour, which feel cystic on palpation; the surface shows patulous pilo-sebaceous orifices and a few follicular pustules. At the lower end of the chief fold there is a little discharge of serous fluid. Subjective symptoms are at present confined to occasional slight itching.

History.—The eruption began at the age of 12 years in the form of itching red spots, attacking chiefly the arms and legs. It spread later to the shoulders and upper part of the chest and back. The face only became involved in June, 1931, and here the eruption began as a boil, which soon discharged a dark brown substance. When first seen by me a few weeks later, the condition of the limbs and shoulder-girdle was as at present, except that there was intense itching. The face was, however, affected by a definite furuncular condition, for which fomentations with biniodide

[1] *Proceedings*, 1929, xxii, 765 (Sect. Derm. 19); *Brit. Journ. Derm. and Syph.*, 1929, xli, 270-273.

of mercury were instituted. This treatment has been maintained ever since, and the cheek has greatly improved, so that there is now very little evidence of infection. A biopsy taken from the forearm reveals masses of interlacing plain muscle fibres in the cutis.

Comment.—Besnier divided cutaneous myomas into two groups:

(1) Multiple myomas: Arising at any point of the skin either from the erectors of the hairs or muscle cells of the blood-vessels. They appear as pink elevations scattered or agminated and reach the size of a peanut or larger.

(2) Dartoid myomas: These are less rare. They only appear where there is a dartoid layer, e.g., mammary areola, scrotum, labia majora. They are often solitary, and may reach the size of a fist.

All myomas may be painful and become harder under the influence of pressure, local irritation or the action of cold, which provoke the contraction of the muscle fibres. There appears to be no successful treatment apart from excision or, in certain cases, electrolysis or diathermy. I cannot find a description of a case with such a very extensive eruption. Itching appears to be an unusual symptom. I do not know of a record of myomas being secondarily infected as in this girl's face. In this region there appears clinically to be an overgrowth of sebaceous glands as well. If the myomas are derived from the erectores pilorum it would not, perhaps, be surprising if other elements of the pilo-sebaceous complex sometimes took part in the overgrowth.

? Pityriasis Rubra Pilaris. Case for Diagnosis.—L. FORMAN, M.D., for Dr. G. B. DOWLING.

J. L., male, aged 60, and six weeks ago noticed swelling of the palms and the soles with some irritation. Later some roughness and scaliness of the neck, front and back of the chest, and the scalp. Seen four weeks ago at Out-Patients' Department. Condition was then as follows: Scalp covered with fine easily detached scales. Forehead and face scaly and a little reddened. Skin on the front and back of the chest and on the shoulders distinctly rough to the touch. There were slight follicular prominences with a little hyperkeratosis of the orifices. On each elbow over the ulna there was a rather sharply defined scaly plaque which was probably due to patient's leaning on his elbows when at work.

The palms and soles were reddish brown, with marked hyperkeratosis and a tendency to fissure along the natural folds. There was an erythematous edge to the hyperkeratotic area. During the past three weeks the patient has been in bed and has improved. The palms and the soles are not so erythematous, and the skin of the chest and the back shows much less follicular prominence. There is a little follicular keratosis of the follicles on the dorsal aspect of the proximal phalanges of the hands.

A section taken from the back showed hyperkeratosis of the follicular orifices and some hyperkeratosis of the skin intervening.

Discussion.—The PRESIDENT asked whether it was clear that the follicular hyperkeratosis was quite recent. [Dr. FORMAN: I do not know.] Clinically it looked more like follicular ichthyosis than pityriasis rubra pilaris, though the history was against that diagnosis.

Dr. SEMON said that he had recently seen a similar palmar eruption in which the diagnosis of lichen planus could be made only on account of a few typical papules on the fronts of the wrists. He wondered if that diagnosis could be applied in this case.

The PRESIDENT said that the thickening of the palms in the present case was not exactly that seen in lichen planus, and he thought that if the lesions on the back were lichen planus they would have presented the typical picture.

Cavernous Nævus treated by Radon Seeds.—J. E. M. WIGLEY, M.B., and R. T. BRAIN, M.D.

A boy, J. A., aged 5 months, attended Charing Cross Hospital with a dome-shaped cavernous nævus on the occipital region of the scalp, two inches in diameter. On July 23, 1931, four silk-threaded platinum radon seeds, each 1 cm. long, content 1·35 mc., were inserted radially, 90 degrees apart, the opposing threads, which emerged on the circumference of the lesion, were knotted together and the whole lesion was covered with collodion. The seeds were removed one week later, giving a dose of approximately 120 millicurie hours from each seed. There was a slight erythematous reaction. On September 3, 1931, the lesion was replaced by a flat, pink, bald area. This is now pale and will be easily concealed by the hair around. Some hair is actually growing on the treated part.

Dr. H. SEMON said he had raised the question of needles versus plaques for the treatment of angiomata in a case shown by the President at the last meeting.[1] The latter method was favoured by the Radium Institute for this type of vascular tumour, although with its frequently delicate epidermal covering there seemed some risk of superficial ulceration in the ensuing reaction period. Dr. Brain had demonstrated that radium needles had a similar effect in promoting involution, and as only one dose had to be given and the risk of surface ulceration was small, this method deserved more consideration than it had hitherto received.

Lupus Erythematosus. (Telangiectatic form).—H. MACCORMAC, C.B.E., M.D.

The patient, a man aged 57, has recently (April, 1931) been operated upon for carcinoma of the stomach. Apart from this there is nothing of importance in the history.

The eruption began on the lobes of the ears in June, 1930, spreading over the scalp and on to the face gradually. Since then the dorsal surfaces of the fingers and hands have become affected.

The interest of the case lies in the form assumed by the eruption on the face and scalp. The scalp is almost universally pink, but without any scarring or desquamation; the skin of the face may be described as florid. This type corresponds most nearly to the very rare telangiectatic form classified by Radcliffe Crocker. It does not suggest, in itself, lupus erythematosus and the diagnosis can only be established by the typical form of the eruption on the hands.

Discussion.—Dr. H. SEMON pointed out that some of the lesions on the neck were lichenoid, suggesting, in his opinion, an erythematous form of lichen planus.

The PRESIDENT said he agreed with Dr. MacCormac, both because of the character of the lesions on the face—particularly those on the forehead—and the type of lesions on the hands. It was like a case he himself had shown at a meeting of the Section some years ago, in which the same kind of eruption extended down the chest, shoulders, and upper part of the trunk.

Acute Lupus Erythematosus with Bullæ.—A. M. H. GRAY, C.B.E., M.D.

Patient, female, aged 33. The rash began with redness of the face after walking in the sunshine in May, 1931. In September, red patches appeared on the backs of the hands and also a large ringed patch appeared on the front of the neck and upper chest. In November the feet became red, and the toes and soles showed a tendency to blister formation. The hands also began to blister and crack, and crusts were formed.

The case is one of unusually acute lupus erythematosus with blister formation on the hands (fig. 1) and feet, and curious ring-like formations with blister edge (which in the case of the first-named lesion had become purulent), on the front of the neck and chest, and on the right side of the neck (fig. 2).

The patient has had no rise of temperature whilst under observation, except on the day of admission. The pulse rate has been rather rapid. The patient has been blind in one eye since birth, and has congenital nystagmus.

Cultures from the blood have proved negative for tubercle bacilli and any other organisms.

[1] *Proceedings*, 1932, xxv, 385-7 (Sect. Derm. 15-17).

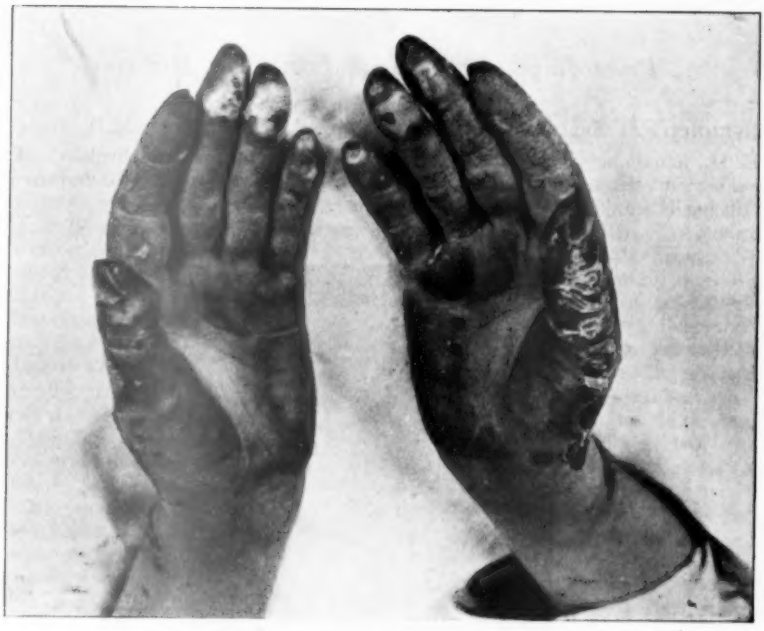


FIG. 1.

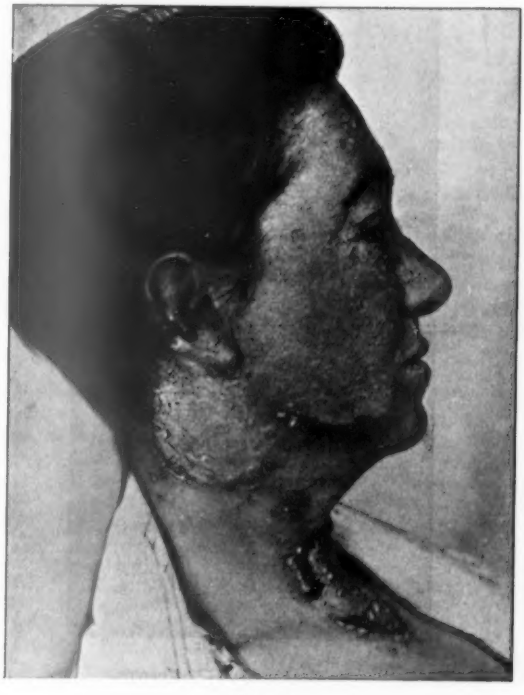
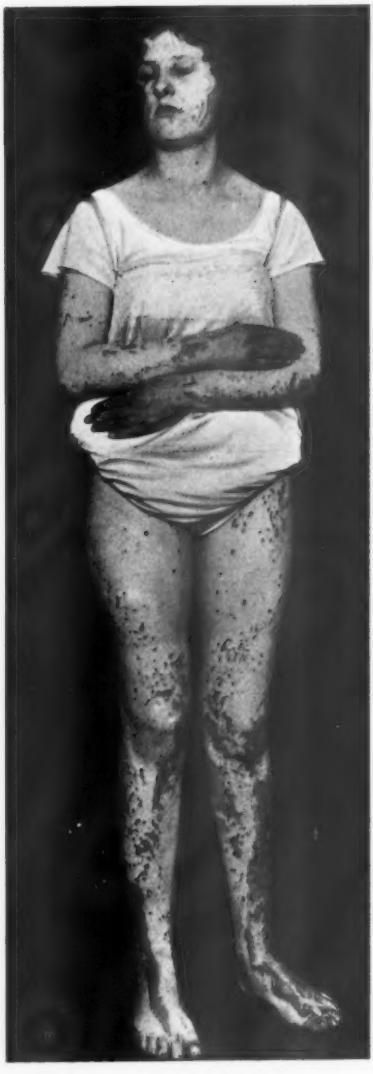


FIG. 2.
Dr. Gray's case of acute lupus erythematosus with bullæ.

Symmetrical Systematized Nævus.—F. A. E. SILCOCK, M.D.

E. H., female, aged 15, only child. Parents both normal and healthy. Patient is of a nervous disposition. Condition of skin first noticed when two months of age first on her left ankle, then on left little finger.



Symmetrical systematized nævus.

There is now an extensive and more or less symmetrical nævus which is polymorphic in character, consisting of pigmented macules, papules, and warty nodules. In colour they vary from light brown to almost black.

Distribution.—The general arrangement is in lines or streaks, which tend to become more marked towards the extremities. These streaks are placed longitudinally on the limbs and neck, whilst those on the chest run parallel to the ribs. *Face and forehead.*—Scattered light brown scaly patches on forehead and both cheeks. *Neck.*—Light brown continuous streak about 4 in. long and ½ in. wide, running longitudinally on mid-line posteriorly. *Chest.*—Scaly patch of very dark brown colour on right side in mid-axillary line, running along the fifth rib and extending over the right breast. *Left arm.*—Lesion runs from shoulder posteriorly down to tip of little and ring fingers in a more or less continuous line. Skin over these two fingers is brown and verrucose. *Right arm.*—This shows a much similar arrangement to that on the other arm, only the condition extends to base of phalanx of index-finger dorsally. *Left leg.*—Scattered brown papules, &c., arranged in streaks from fold of groin to bases of great and little toe on outer side. Posteriorly they extend from buttock in mid-line to back of heel. *Right leg.*—A similar configuration is shown here to the other leg, except that anteriorly it extends to base of the second toe dorsally. *Heels.*—These both display marked rough tylosis formation and tylosis is also found under the ball of the right great toe.

The case is brought forward owing to the extent and symmetry of the lesions; also because it is somewhat uncommon to find them on the face and forehead as well as on the rest of the body.

Discussion.—Dr. H. MACCORMAC said he believed this to be an example of porokeratosis of Mibelli. It presented in characteristic form many of the features of this very rare disease, viz: the gradual onset, the barrier-like edge, and the reddish patches upon which milia-like bodies are observed. The peculiar plantar hyperkeratosis was further in favour of this view.

Dr. SEMON agreed with Dr. MacCormac that there was marked similarity in the type of eruption with that under their observation at the Base Hospital in France in 1917. Dr. MacCormac would remember that the eruption in his case was unilateral, involving the shoulder and upper extremity only, and to the best of his recollection had not been present for more than five or six years.

Dr. PARKES WEBER thought the case was undoubtedly one of bilateral systematized nævus (of the type of a *nævus unius lateris*, but affecting both sides of the body). The structure of the nævus was hyperkeratotic or ichthyosiform on telangiectatic areas, with some pigmentation. Dr. Weber thought it was very unlike the descriptions of porokeratosis Mibelli.

Dr. GOLDSMITH said that in Darier's textbook there was an illustration of a linear nævus similar to the lesions in the present case, bilateral and symmetrical. He thought that porokeratosis developed as a circular wall and not in straight lines, and that the hyperkeratotic walls were very much sharper.

Poikiloderma (Civatte) following Toxic Erythema.—J. E. M. WIGLEY, M.B. and R. T. BRAIN, M.D.

H.L., a widow, aged 53, has suffered from chronic paronychia, and about nine months ago, erythematous eruption developed on the face, forearms, and upper sternal region, with considerable œdema of the eyelids. The onset was sudden, accompanied by malaise, sore throat and constipation. She had a slightly raised temperature, with pain in the larger joints and abdominal tenderness. No definite cause was discovered for the abdominal condition, and there was no evidence of nephritis. As the condition persisted, she was admitted to Charing Cross Hospital for further investigation, in July, 1931. She was found to be well nourished, but apparently too weak to sit up in bed without assistance and she complained of abdominal discomfort. There was œdema of the face about the eyes, and scaly erythematous patches were present on the forehead, sides of the face, neck and

upper sternal regions and between the shoulder-blades, on the extensor surfaces of both arms, on the buttocks, the thighs and the soles of the feet. A moderate fever (100°F.) persisted. The blood-pressure was 126/70. The heart was not enlarged, and no signs of disease were discovered in the respiratory or central nervous system. The tongue was furred, and there was fœtor oris. Tonsils enlarged and inflamed, teeth good, but there was severe gingivitis. The abdomen was slightly distended and generalized tenderness was complained of, and there was enough rigidity maintained to make palpation futile. A surgical colleague saw her, but could find no cause for interference. The urine contained a trace of albumin; there were no cells and it was sterile on culture. Four blood-counts were made. They showed little variation and a typical one is as follows:

R.B.C. 4,100,000; Hb. 76%; C.I. 0·9; W.B.C. 6,800. *Differential*: Polys. 51%; eosinos. 5%; lymphos. 33%.

An X-ray examination of the chest revealed no pulmonary lesion, but the right dome of the diaphragm was high and a suggestion of a neoplasm or hydatid cyst was made. The complement deviation test for hydatid was negative. Two further skiagrams showed a normal size gall-bladder and the same peculiarity of the diaphragm. The patient was two months in hospital and during that period the erythematous areas lost their scales and became of a dull reddish brown tint. She was sent to a Convalescent Home and when seen again on November 12, 1931, the pigmentation was the prominent feature.

A brown pigmentation covers the forehead, sides of the face, neck and exposed portions of the chest. It is also present on the backs of the hands, wrists and forearms. The shade of the colour varies; in places the pattern is definitely reticular, and a few small telangiectases are present. A very slight degree of atrophy is visible in places.

The patient states quite definitely that the presence (or absence) of bright light has made no appreciable difference to the eruption, and the fact that the pigmentation developed during her stay in hospital makes that factor very improbable. The combination of reticular pigmentation, telangiectasia and fine atrophy, occurring on the sides of the face in a woman about the time of the menopause, corresponds so closely to the description given by Civatte that we have ventured to give this case the title he suggests.

Discussion.—Dr. PARKES WEBER suggested that the case might be one of Jacobi's poikilodermia (atrophicans vascularis) rather than one of Civatte's poikilodermia—as there was much alteration of the texture of the skin in some parts other than the face.

Dr. BRAIN in reply said that the condition poikiloderma was not so well defined that cases could be separated into two groups. The telangiectasia and atrophy were difficult to recognize in all cases, and as one often saw minute spots of atrophy on the skin of elderly women, the atrophy might not be an essential feature of poikiloderma. Civatte described a reticular mottling in the distribution. This was present in the case now shown.

Section of Epidemiology and State Medicine.

[*January* 22, 1932.]

Some Aspects of Industrial Medicine.

By N. Howard Mummery, M.R.C.S., L.R.C.P.

It seems necessary first to define what is meant by "industrial medicine," and I would describe it as concerned primarily in assisting industrial management to maintain an efficient personnel by applying preventive and remedial means in reducing to a minimum the working time lost by employees owing to illness and injuries.

This is not the time to enter into any retrospective consideration of the subject, but you be well aware that, although the scientific application of medical knowledge to industrial management is of recent origin, the protection of workers against industrial hazards dates back to pre-Roman days. Yet little over a century ago public opinion in this country tolerated such appalling industrial conditions as the requisitioning by manufacturers of child labour from workhouses and the insanitary "bothies" in which these wretched infants, regardless of sex, were locked up at night. The past twenty-five years have seen the pendulum swing almost to the other extreme, and with National Health and Unemployment Insurance, Free Education, Employer's Liability and Workmen's Compensation Acts, innumerable Home Office Regulations and Welfare Orders, in addition to hospital and numerous other philanthropic health services, we find the industrial workers supported on every side by social props, largely financed by other sections of the community who do not share the benefits, and we see clear evidence that they are losing their healthy spirit of independence and neglecting to make provision for themselves and their dependants. Probably the financial burden of these social services has fallen most heavily upon the employer of labour, and viewing the matter from his angle, one might well think that there is a general conspiracy on the part of the State, the National Health Insurance Administration, and even of many insurance medical practitioners and certifying factory surgeons, to load the employer with responsibility for all the ills that beset his workpeople. And this in spite of the well-recognized fact that skin diseases, herniæ and varicose veins—to mention only three common examples—are not peculiar to the industrial section of the community, and actually are less likely to arise in a properly conducted and hygienic factory than they are in the homes of the people.

I propose, therefore, to confine my remarks to that aspect of my subject which concerns industrial management.

Returning to the employer's viewpoint, it will be obvious that his main concerns must be to secure a reasonably healthy staff, and to obtain from his workpeople a maximum amount of efficient labour with a minimum of absenteeism. He finds that the engagement of personnel without vocational selection and without medical examination tends greatly to increase the claims made upon him in respect of accidents and alleged injuries, and he finds that dependence solely upon medical certification of incapacity for work by practitioners who have no direct interest in curtailing delayed recovery or malingering, tends to an undue amount of absenteeism. Various solutions to his difficulties in these respects have been tried, and the method that is proving the most satisfactory in large concerns is also proving humanitarian. This consists, briefly, in supplementing the existing State services, payments, and

facilities for treatment, but at the same time providing such help under close medical supervision by a medical practitioner, or Works Medical Officer, whose services he retains in either a whole or part-time capacity.

Published figures for 1930 show that in this country there were approximately 142,000 factories and 133,000 workshops, large and small. Of these, 2,200 of the larger concerns, 88% of which are factories, have adopted supplementary health services. Details obtained from 1,006 of these concerns show that between them they employ 2,022 nurses, most of whom work under medical supervision of some sort, and 91% of these firms employ doctors on a full or part-time basis. (*Public Health Nurse*, March, 1930.)

The duties of the industrial practitioner are manifold, but mainly they consist in primary examination of candidates for employment to insure, on the one hand, that the work proposed will not aggravate any existing disability, or light up a pre-existing state, and, on the other hand, to protect the employer against false claims at a later date, in respect of such disabilities or physical tendencies. Thereafter his duties consist in adopting every possible preventive and remedial means to reduce absenteeism due to illness or injuries, and it is interesting to note that as accidents are largely a "reaction to environment," common also to most sickness, the incidence rates for both injuries and illness fall or rise together as the speed of the work and the environment alters. The prevention of absenteeism, apart from the hygienic questions of ventilation, heating, lighting, baths and general sanitation, largely consists in encouraging employees to attend the Factory Clinic when the earliest signs of illness are noticed, so that suitable treatment may abort the trouble or a few days' absence and treatment may prevent more serious complications, and to report immediately all accidental injuries or commencing skin disease, however trivial, when prompt treatment may enable the employees to continue work and prevent the occurrence of sepsis or secondary troubles. The following figures in connection with Messrs. J. Lyons & Co.'s Cadby Hall Clinic, of which I am in medical charge, are of interest:—

During 1930, 9,995 injuries were treated at the Clinic; of these 8,455 employees remained at work, and 1,540—representing 15·4%—were put off duty; 12,748 re-dressings were done, 11,538 of these were during the continuance of employment, and 1,210—representing 9·5%—while away from work; there were 2,255 attendances of skin cases of all kinds, 1,797 of these while at work, and 458—representing 20·3%—during absence from work. (In this connection I insist upon a short absence from work in all acute skin diseases because I find it considerably shortens the duration of the complaint and ultimately prevents loss of time.) 12,258 cases of illness were seen and treated, 9,446 of these remained at work, and 2,812—representing 23%—were sent home; 2,415 persons were medically examined for employment; 1,033 patients from outlying hotels, corner houses, and tea-shops were seen in consultation.

I have tabulated the various causes of absenteeism amongst the factory operatives during 1930, showing the number of cases amongst males and females respectively, the time lost, and the average duration of absence in each case. It should be noted that amongst these operatives the males exceed the females in number by about 2,000. This table shows that acute respiratory diseases account for the largest number of cases, affecting the sexes fairly equally, there being 1,157 cases in all, with a total of 12,510 lost days, giving an average of 11 days per case. Acute gastritis and gastro-enteritis comes next in frequency, affecting females much more than males, with a total of 504 cases and 4,615 total days lost, giving an average of 9 days per case. Fibrositis was more prevalent amongst the males, the total number of cases being 261, and the total days lost 4,368, with an average of 17 days per case. Diseases of the ear, nose and throat were more common amongst the females, with a total of 265 cases, and 3,189 lost days, giving an average of 12 days per case. Furunculosis was about three times as common among the males as

FACTORY STAFF. JANUARY 1 TO DECEMBER 31, 1930.

Average Personnel: Males, 3,324; *Females*, 1,461. *Total*, 4,785.

	Males		Females				Days per case
	Cases	Days lost	Cases	Days lost	Total cases	Total days lost	
Respiratory Diseases—							
Acute	666	7,326	491	5,184	1,157	12,510	11
Chronic	2	22	2	32	4	54	13·5
Pulmonary tuberculosis	2	12	3	168	5	180	36
Digestive Diseases—							
Acute gastritis, enteritis, colic	205	2,232	299	2,383	504	4,615	9
Appendicitis	17	774	14	737	31	1,511	50
Peptic ulcer	11	563	1	26	12	589	49
Chronic gastritis, etc.	4	146	1	7	5	153	30·5
Other forms, jaundice, teeth	26	485	37	318	63	803	13
Rheumatic Diseases—							
Rheumatic fever Chronic arthritis	4	134	2	63	6	197	33
Fibrositis, myalgia, lumbago, etc.	169	2,338	92	2,030	261	4,368	17
Circulatory Diseases—							
Heart	11	505	3	85	14	590	42
Vessels	14	843	4	43	18	886	49
Anæmia	8	101	48	656	56	757	13·5
Apoplexy and other disorders	2	166	—	—	2	166	81
Acute Infectious Diseases	36	567	52	642	88	1,209	13·5
Diseases of Ear, Nose, Throat	120	1,559	145	1,630	265	3,189	12
Diseases of the Eye	30	429	14	146	44	575	13
Diseases of Skin—							
Furunculosis	99	1,275	29	373	128	1,648	13
Other dermatoses	50	945	45	791	95	1,736	18
Nervous Diseases—							
Organic, mental, epilepsy	5	253	2	30	7	283	40
Functional, neurasthenia, etc.	14	401	25	399	39	800	20
Diseases of nerves, sciatica, neuritis, etc.	46	614	60	444	106	1,058	10
Genito-urinary Diseases (dysmenorrhœa)	9	190	188	1,155	197	1,345	7
Other Diseases—Malaria, etc.	14	213	44	571	58	784	13·5
	1,564	22,093	1,601	17,713	3,165	40,006	12·7
Injuries—							
Non-fatal	660	8,805	226	2,732	886	11,537	13
Fatal	—	—	—	—	—	—	—

among the females. There were 128 cases, and 1,648 lost days, with an average of 13 days per case. Neuritis predominated among the females, but the males, on account of the larger incidence of lumbago and sciatica among them, were longer away from work than the females. The latter, on the other hand, suffered more from brachial neuritis, which, however, cleared up more quickly. There were 106 total cases, with 1,058 days lost, and an average of 10 days per case. Genito-urinary diseases were almost entirely confined to females. Tubular disease, uterine displacement and true dysmenorrhœa accounted for most of the cases, the total number of which was 197, and the total days lost 1,345, giving an average of 7 days per case. Very few cases of venereal disease in either sex occurred. Taking into account the number of males and females employed as factory operatives, the incidence of accidental injuries was fairly equally distributed between the sexes, and the average of 13 lost days per case from this cause compares favourably with published statistics.

The incidence of illness is noticeably more among the females than the males. The 1,461 female operatives provided 1,601 cases, being 109·58%, whereas among the 3,324 male operatives there were 1,564 cases, or 47·05%. The duration of sick absence, on the other hand, was more prolonged among the males, being 22,093 days, or 14·12 days per case, as compared with 17,713 days, or 11·06 days per case, for the females. This agrees with the Government Actuary's Report on the Third Quinquennial Valuation of Approved Societies, wherein he states that the sickness claims of women are everywhere in excess of those for men, but it is not in accord with his statement that women workers remain longer in insurance. In

estimating future insurance claims, the Actuary has been compelled to allow 25% more weeks of sickness benefits and 65% more weeks of disablement benefit for single women than for men, and for married women 140% more sickness benefit and 60% more disablement benefit, than for men, and he states that all investigations show a higher sickness-rate for women than for men in industry. In an editorial article on this report the *Lancet* (1932 (i), 89), asks whether welfare work and improved factory hygiene are preventing this excess of sickness among women, and I think my figures show that medical supervision in industry is, in fact, largely reducing the excess sick absenteeism to which the Government Actuary refers.

Sir George Newman's recent report showed that the insured population of England and Wales lost on an average 3·73 weeks' working time per annum, and Sir Thomas Oliver (*Journal of State Medicine*, August, 1930) states that during the

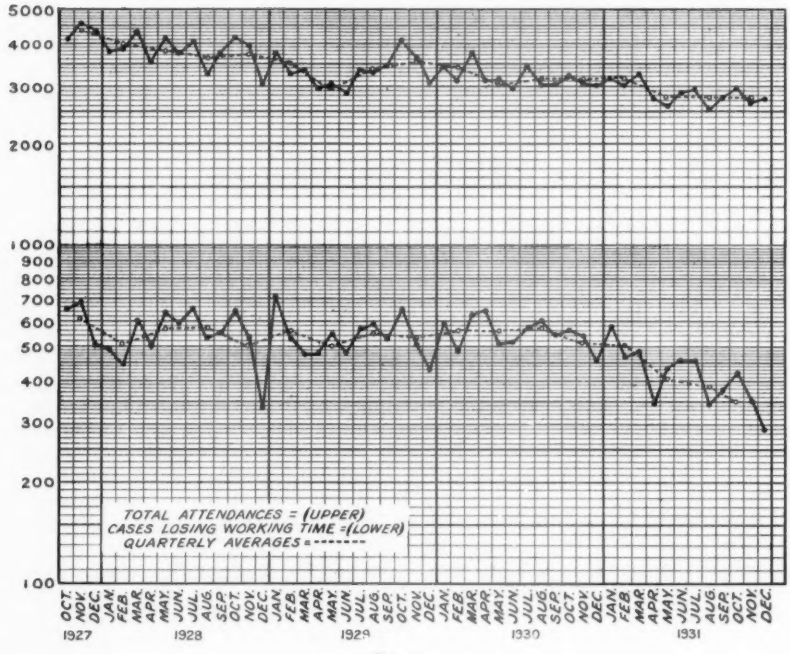


FIG. 1.

past thirty years the length of sickness has increased from 16½ days to 28 days per annum. It is interesting to compare with these figures for the insured population of the country as a whole, those of my Clinic, which are as follows. The factory staff lost on an average 13 working days each in 1929, and 8 days each in 1930. The average duration of absence on account of illness in 1929 was 15 days, and in 1930 it was 12·7 days. The average duration of absence on account of injuries in 1929 was 13·3 days, and in 1930 it was 13 days.

In order to demonstrate the practical results of medical supervision in industry, I have prepared a chart on logarithmic ruling covering the past 4½ years (see fig. 1).

This shows the monthly fluctuations and quarterly averages in total attendances at the Clinic, and also the number involving lost working time. It will be seen that, with marked rises and falls during the intervening years, the total attendances with an approximately comparable number of employees have fallen from 4,563 in November, 1927, to 2,695 in November, 1931, and that the proportion of those losing working time has shown a similar decrease from 635, or 15%, to 325, or 12·75%, in the same period. I mention the month of November because I wrote this before the December figures had been worked out, and it will be seen on the chart that in the latter month there was a sharp drop in the number of those losing working-time—down to 293, or 10·77%—although total attendances remained on much the same level. This was probably due to a tightening up of supervision over absent employees, necessitated by the national crisis in industrial affairs. Unfortunately my figures for the present month of January are, as usual, showing the effect of the influenza epidemic. The fluctuations in the total attendances are governed by various factors, not, unfortunately, due only to the incidence of injuries and illness as, particularly in October, 1929, an unusual number of patients come up in certain months for special examinations. The ratio of absenteeism, or patients put off duty out of the total attendances, tends to be higher during the summer months, with the exception of the month of January which had the highest figure for the years 1929 and 1931. I attribute the very satisfactory reduction in the number of employees losing working-time mostly to the fact that injuries, with few exceptions, are treated throughout by me at the Clinic where an X-ray installation, massage and remedial treatment are available. The duration of sick absence is mainly governed by panel certification over which, naturally, I have little control, but, as shown by the satisfactory and steadily declining average duration of such absenteeism, much can be done by co-operation with insurance medical practitioners and by providing assistance in obtaining hospital and convalescent treatment. We have our own contributory scheme which covers the cost of in-patient maintenance and out-patient attendance at the West London Hospital, under a voucher system. Not the least important factor in controlling unnecessary absence from work is the system my firm has in force whereby the difference between panel benefits and full wages is made up to employees during sickness subject to length of satisfactory service and to my reports on each case—the patient, unless in hospital or confined to bed, coming to see me each week to report progress.

The subject is a large one, but I think I have said enough to show that industrial medicine can render very real service of cash value to the employer, and that in making use of such schemes of defence, industrial management is giving evidence both of good business acumen and good citizenship, for unless the business concern is able to earn good profits, it can neither pay good wages nor extend its activities to provide employment for increasing numbers of workpeople.

At the present time, British manufacturers, with an almost unbearable load of taxation, are fighting to regain lost trade and extend employment, and it behoves our profession to give them all possible assistance. This, I submit, can be done by endeavouring to eliminate the common causes of industrial absenteeism rather than by propounding scientific theories regarding physiological problems. And I would plead for closer co-operation in this work between the scientific members of the Industrial Health Research Board and the physicians of large industrial concerns, who are in touch with the actual working conditions. The common causes of absenteeism are indicated by the plan showing the relative incidence of diseases in my firm during 1930 (see fig. 2, p. 30). In this plan, fibrositis covers the disease group usually attributed to, and certified as, rheumatism. Actually, during the year under review there were only six cases of rheumatic arthritis and none at all of acute rheumatism. The rest of the cases were painful affections of fibrous and connective tissue and therefore, in my opinion, best classified as fibrositis.

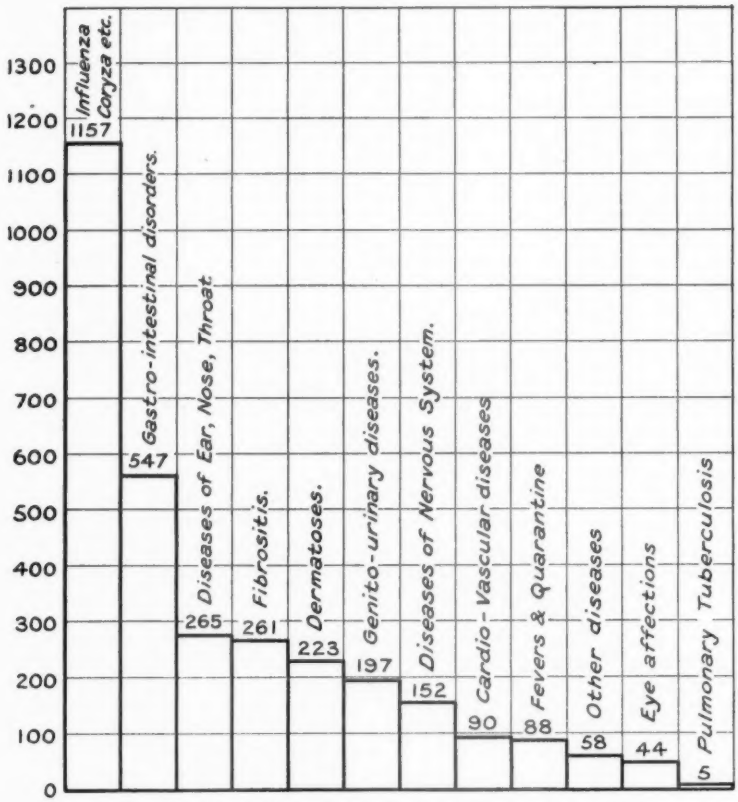


FIG. 2.—Relative incidence of diseases, factory personnel, year 1930.

Discussion.—Dr. H. M. VERNON said that he was specially interested in the graph indicating the absenteeism due to the sickness of the whole body of factory workers between the years 1927 and 1931, for this graph showed a gradual and substantial fall. Such an experience was very different from that observed in the majority of industrial workers, for the Chief Medical Officer of the Ministry of Health had shown that the total sickness and disablement benefit of all insured workers between the years 1921 and 1927 rose 65%, and it was still rising. The study of the numerous psychological, economic and other factors concerned in the variations of sickness absenteeism was most important, if we were to do anything to reduce it from its present very high figure. Several pieces of evidence on the subject had been obtained from time to time by the investigators attached to the Industrial Health Research Board. For instance, he (the speaker) had investigated the sickness of 20,000 iron and steel workers for a six-year period before and during the war, and he found that in the war years the men lost 31% less time than in the pre-war years, presumably because they were "keen to do their bit." In company with Dr. Bedford he had investigated the sickness and accidents of 23,000 coal miners for a two to six-year period, and had found that when the men had their scale of wages reduced, and were to some extent put on short time, their sickness absenteeism fell 25%. Presumably this was because they could not so easily afford to claim benefit. Again, it was found that the men working in deep and hot pits lost considerably more time from accidents than men working in cool and shallow pits; but when

the accidents were classified according to their severity, it was found that severe accidents, entailing disablement for more than ten weeks, were just as numerous in the cool pits as in the hot ones, whilst minor accidents, involving less than ten days' disablement, were four times more numerous in the hot pits than in the cool ones. Probably the accidents incurred were about the same irrespective of the temperature, but when the men working in hot pits incurred a minor accident they were much more liable to claim compensation than the men working in cool pits, as they had a lively recollection of the unpleasant conditions of work. When they incurred a severe accident, however, they had no option but to go on compensation whatever the conditions under which they happened to be working. Evidence relating to another industry, namely weaving, had been obtained by Dr. A. B. Hill. He found that married women weavers claimed more than twice as much sickness benefit as the unmarried women, probably because the married women had many more home and family ties, rather than an actual excess of sickness. Again, Hill examined in detail the day of the week on which illnesses began and terminated, and he found that in printers more than half the illnesses conveniently ended on a Saturday, thereby enabling the men to return to work afresh on the following Monday. The question arose as to whether panel doctors were not apt to grant sickness certificates too freely, without proper medical examination.

Dr. SIBYL HORNER said that Dr. Howard Mummery had mentioned the requirements of the law for safeguarding the health of workers. She would like to add a few words on this subject as it affected women workers. The legal protection of women workers in Great Britain was of especial interest, for in this we were much in advance of other countries. It was important to bear this fact in mind when reading accounts of ill health among the women in industry in countries other than Great Britain. Some of these accounts, authentic though they must be, read like the more sensational of the Sunday press to those acquainted with industrial medicine in this country. Some of the credit for the better condition of the health of women workers in Great Britain must be given to protective legislation. Protection by this means was accorded to minimize the risks in the trades designated as "dangerous"; from some of these women were rigidly excluded—in others work was permitted in the less dangerous processes. Apart from legal restrictions directed against the risks of industrial poisons, women were protected in the pottery and woollen and worsted industries from the lifting of loads that might prove injurious.

Such, with the addition of certain restrictions of hours of work, was a very brief outline of the special protection afforded by the law in the interests of the health of industrial women. There were, she (the speaker) knew, two schools of thought as to the ultimate economic effect of such restrictive legislation, but comparing conditions in this country with those of some other countries, we had, she submitted, no cause to regret the provisions of the law as to women's employment.

Dr. MILLAIS CULPIN said that a survey of industrial sickness absenteeism revealed many vagaries. Days lost per head per annum varied from 3·8 to 15 or 16. In comparable establishments the differences might be extraordinary and it was disconcerting to find that one of the highest rates was in a firm that was recognized as doing its best for the hygiene and general welfare of its employees: it would not be fair to indicate the firm.

Examination of diagnoses showed that, between comparable firms, a difference in the total was often made up of disorders loosely called "nervous." In printers, from 30% to 40% of all days of disablement was due to "disorders of the nervous system"—a group that, in this case, included nervous debility and cerebral hæmorrhage! In an establishment whose records for some years were available to him, the loss from the "neuro" group so alarmed the medical officer that he set out to reduce it. In this he succeeded, but the total remained the same, for the "gastric" group grew at the expense of the "neuro." There was a group of very dubious diagnoses that accounted for excess of lost time, and examples of these could be found in the figures before them that night.

"Acute gastritis, enteritis and colic" were seen to cause a loss of 4,000 days, with an *average* duration of nine days. If he had a case of one of these conditions which, under treatment, did not end in death or recovery within a week, he should think of poisoning by arsenic or lead.

Neuritis was a very common industrial diagnosis, but in 1,000 consecutive cases sent from the London Hospital receiving room to the out-patients department, there was, besides sciatica, only one case marked "? neuritis." Dr. Mummery had referred to cases of brachial neuritis among women. That was a rare disease if one postulated that one must specify the

nerve that is neuritic. "Fibrositis" was a recent discovery and when used as a synonym for myalgia—an old army friend—covered a multitude of very doubtful conditions.

On the other hand, the loss from the "neuro" group was only 400 days which, when the average worked out to 12·5 days per head per annum, was too small. He thought that many cases from the heavier groups should find their proper place here.

Epidemiologists now had to study morbidity statistics, and they had seen to-night some of their pitfalls. The root of the evil was the compulsion upon the panel practitioner to give a name to every disability. He should be encouraged to use something like the army N.Y.D. or the more homely G.O.K.

Fleet Surgeon W. E. HOME said that it was always difficult to name correctly the slight diseases seen from day to day. It was pleasant to have heard from the speakers at this meeting, and to read from year to year in the *Annual Report of the Chief Inspector of Factories to the Home Office* how much was being done for the welfare of the workers in industry. There were 899 deaths from injury in the factories in 1930, and the Home Office spent fifty pages in telling what had been done to prevent them in future. On the other hand, the Board of Trade took no means of letting them know what they did to prevent the repetition in future years of the 400 deaths from injury which they reported among the 150,000 British seamen in 1929.

Dr. D. A. COLES drew attention to the great diversity of methods adopted by different employers of labour in permitting employees suffering from pulmonary tuberculosis to resume work. He referred to a particular instance of a business in which a very large number were employed, where the custom was to permit the workers to resume employment even though the disease was active and tubercle bacilli were found in the sputum.

Men employed in hard work experienced less sickness disability than the average individual. He emphasized the important rôle of the Home Office in combating industrial disease and particularly in helping the industrial doctor.

Dr. J. D. ROLLESTON wondered if Dr. Mummery had any experience of diseases prevalent among workers in tobacco factories. Among other causes alleged to account for the recent increase in primary carcinoma of the lung, the dust in these factories as well as the great increase in smoking had been incriminated (Lickint[1]). Employment in tobacco factories was also supposed by some to render pregnant women liable to abortion (Gy[2]).

Dr. G. CLARK TROTTER said that in Islington he had endeavoured, by taking an interest in the welfare work of a number of firms, to stimulate this work and to put it forward as an example to other firms. He was firmly convinced that welfare work was well worth while, not only from the point of view of the individual worker who came under its influence, as those firms which had adopted it found that it paid in various ways. In consequence of better attention to the employees, a higher standard of efficiency was obtained, and they attracted on to their staffs a better class of employee. It was to be regretted that welfare work was not more widely adopted.

The Factory Welfare Orders undoubtedly, so far as they went, were only productive of good, and were the starting point of much improvement. For instance, before the Welfare Orders for workers in bakehouses, biscuit bakers and confectioners, it was extremely difficult to get anything like a minimum of "decent" ablution fittings in factories and workshops. The excuse often made by the master baker was that it was not expedient to provide soap, as it might happen to get into the "batch." Now, although improvement comes slowly, there was an urge to provide it, and some firms who stood out had spent considerable sums and had provided efficient accommodation. There was, however, one section not coming under the heading of "factories and workshops" in which there was a crying need for better welfare—he referred to shops in general. In an investigation in Islington of the general welfare arrangements, not one firm had these arrangements or a woman welfare supervisor employed. In about one half (46·6%) an ambulance box was provided.

Generally speaking, it could not be said that the proprietor was indifferent to the health of the employee; in about half the places visited it was found that some interest was taken. The interest was greater in the smaller type of shop, and tailed off in the very large shops. Some large firms showed very real interest in their employees, but among the rest, as the size of the business increased, the personal touch and interest in the employees waned. Some of the large multiple shops were the most inconsiderate in this respect.

1 Lickint, F., *Zeitschr. f. Krebsforsch.*, 1929-30, xxx, 361.
2 Gy, A., *Thèses de Paris*, 1908-9, No. 237, 235.

Section of Ophthalmology.

CLINICAL MEETING, HELD AT THE ROYAL EYE HOSPITAL, SOUTHWARK, ON DECEMBER 11, 1931.

Retinal Detachment—Gonin's Operation with Foster-Moore Stud.—Sir RICHARD CRUISE, K.C.V.O., F.R.C.S.

E. M., aged 68. Nothing of interest in history till 1926. V.R. and V.L. = $\frac{6}{9}$ with $-2{\cdot}5$ D. sphere.

3.10.1931.—The vision blurred, following a cold, and for one week patient has seen the tops of objects only. L.V. = counting fingers at 6 in. Vision too poor to take a field with 4° white object. Large detachment upwards and outwards.

7.11.31.—Shallow detachment of almost whole retina. Hole seen at 10 o'clock 12 mm. from limbus.

11.11.31.—Cautery puncture after use of Foster-Moore stud.

10.12.31.—L.V. = $\frac{-\ 2{\cdot}25}{-\ 1{\cdot}0} = \frac{6}{36}$. 1° white field almost full except for slight upper nasal loss. No detachment seen, hole closed.

A fair-sized hole was discovered in the extreme periphery, upwards and inwards. Having mapped it out from 11 mm. to 12 mm. back, I used a Foster-Moore pin. This is the first time I have employed that aid and I found it very useful. In this case the pin was 1 mm. too far back, and I allowed for this error in the cautery puncture. There is now no detachment. There is a clear scar where the retina is adherent, and the vision has improved from perception of hand movements to $\frac{6}{24}$ partly.

Metastatic Carcinoma of the Choroid.—O. GAYER MORGAN, F.R.C.S.

This patient, a woman, aged 36, had a carcinoma of the breast, which was left too long unoperated on and had to be treated with radium. Three months ago she complained of failure of vision in one eye, and there is a central detachment of the retina. It is not a balloon-shaped detachment, it looks solid and extensive, and there are no new vessels to be seen on it. I think it is a metastasis in the choroid, from the original breast tumour. The other eye is healthy.

Retinitis Punctata, ? Albescens.—J. F. COLE MARSHALL, F.R.C.S.

Such cases as this are uncommon. I have no doubt about the diagnosis, but in the literature on the subject it is generally stated that in this type of case, vision is maintained at practically the same degree of acuity. Mr. Nettleship showed three cases in 1885, and brought out the point that the condition was stationary. The alarming feature about this patient is that since February, 1931, his vision has decreased from $\frac{6}{12}$ to less than $\frac{6}{60}$, and at the macula there is a fine retinitis. In 1926 he had many bad teeth extracted and afterwards his vision improved. Taking into account the fine nature of the pigment at the macula, I think two things are present: retinitis punctata albescens, and a fine retinitis, due to some septic condition. The remainder of the teeth are to be extracted, and I shall note whether the vision improves afterwards.

Discussion.—Mr. CYRIL WALKER asked whether in cases of true retinitis punctata albescens, marked night-blindness was not almost always present. This patient had little night blindness.

The PRESIDENT said this patient had told him that he had night-blindness, but that as he was an engineer, he was able to cope with it more easily than the average person could do.

Opacity of the Cornea.—H. M. JOSEPH, F.R.C.S.

This apparently healthy youth, aged 17, has a corneal opacity stretching from the limbus to the centre. During the last six months this has gradually increased. There has been neither pain nor redness, and the history is uneventful. I shall be glad to know what it is, and how to treat it. The Wassermann reaction is negative.

The PRESIDENT said that the opacity had begun below, and spread upwards without causing pain. He thought that a fungus existed beneath the epithelium, and worked its way along Bowman's membrane, and that what was now seen indicated the limits of the growth. His advice was to scrape the opacity away with a small spoon.

Nævus of Conjunctiva.—J. D. MAGOR CARDELL, F.R.C.S.

Mrs. M. G., aged 34, has nævus of the right side of the face and nose, involving the caruncle and the palpebral and bulbar conjunctiva of the nasal half of the right eye. The condition has been present since early childhood, and has shown no appreciable increase. R.V. with $-1{\cdot}0$ DC., $\frac{6}{6}$; L.V. with $-8{\cdot}0$ DS. $\frac{6}{6}$. Both fundi are normal. The skiagram of the skull is normal. This case may well form a connecting link between cutaneous nævus of the face and nævus of the retina and choroid.

Mr. A. W. ORMOND said that this probably fell into the group of cases about which Ballantyne had written in the *Journal of Ophthalmology*, in which there was a nævoid condition of the skin associated with glaucoma or buphthalmos, or sometimes with a defect in the visual fields. These associations with congenital lesions interested him very much, because it was unusual to encounter a patient with only one. The note stated that the left eye was larger than the right, the right eye being hypermetropic and the left eye myopic, and he thought that probably this was a case in which there had been some increased pressure in the left eye in early life.

Punctate Keratitis. ? Nature.—J. FOSTER, F.R.C.S.

J. W., male, aged 23. Nothing of interest in past history or on general examination except a positive intradermal tuberculin reaction.

Right cornea.—On slit lamp examination there is seen an irregular circle of round spots of infiltration $0{\cdot}5$ mm. diameter, situated at varying depths in the parenchyma. The more superficial of these have ulcerated and stain faintly. Pannus and epithelial œdema. Slight ciliary injection. No disturbance of sensation. No keratitic precipitates. Vision $= \frac{6}{6}$.

Left cornea.—Nebulæ with similar arrangement in the parenchyma of the cornea. Slight pigmentation, ? phlyctenular. Vision $= \frac{6}{6}$. Condition has slightly improved since removal of foul teeth.

Right and Left Endothelial Dystrophy of the Cornea.—CHARLES GOULDEN, F.R.C.S.

J. S., female, aged 58.

On November 27, 1921, complained that her left eye had been misty for a few weeks, and that the right eye was also affected, but less than the left. Pupils equal and active, tension normal.

R.V. $\frac{6}{12}$ not improved, $+ 2{\cdot}7$ 5D. = J2. L.V. $\frac{6}{12}$ not improved, $+ 2{\cdot}7$ 5D. = J2.

Eyes not injected; no corneal precipitates. When examined with the ophthalmoscope the view of the fundus is very misty. No lesion found in either fundus. The mistiness is due to changes in the posterior surface of the cornea. Examined by the slit lamp, the endothelium has a bronzed appearance. There is loss of outline of the endothelium, and there are many round, black, non-reflecting surfaces in the zone of specular reflection. By transillumination the appearance is as if the posterior surface of the cornea had been covered with a fine spray of oil.

Separation of Berger's Layer of the Lens Capsule.—CHARLES GOULDEN, F.R.C.S.

H. B., male, aged 69, chef.

History.—Right eye has failed during last four years. Left eye has not markedly changed. He has been working at a grill for forty-seven years and he thinks that both eyes have been equally exposed to the heat of the fire.

Right eye.—Not injected. Pale atrophic iris. Posterior synechiæ. A good deal of pale corneal precipitate with endothelial bedewing. When the pupil is dilated

it will be seen that the zonular layer of the anterior lens capsule has become detached and protrudes as a folded transparent membrane into the anterior chamber, almost touching the cornea. There is a good deal of uveal pigment on the anterior lens capsule. R.V. $\frac{6}{24}$ + 3·00 D. sph. $\frac{6}{12}$, + 6·00 D. sph. = J1. Lens clear. Vitreous opacities. No fundus lesion.

Left eye.—Iris brown. Scattered iris pigment on posterior surface of cornea. Lens clear; capsule unchanged. L.V. $\frac{6}{18}$ + 2·50 D. sph.
+ 1·00 D. cyl. 90° = $\frac{6}{9}$ + 3·0 D. J1.

Vitreous clear. No fundus lesion.

Glaucoma Capsulo-Cuticulare.—CHARLES GOULDEN, F.R.C.S.

E. S., female, aged 67, housewife.

9.7.29.—Came to Royal London Ophthalmic Hospital complaining of some defect in vision, right eye.

R.V. $\frac{6}{18}$ J8, + 1·50 D. sph. $\frac{6}{12}$ + 3·00 D. sph. J4.

L.V. $\frac{6}{18}$ J12, + 1·50 D. sph. $\frac{6}{12}$ + 6·00 D. sph. J4.

Right tension noted full normal. Left tension normal. Right and left lens opacities.

30.10.29.—Right and left vision reduced to $\frac{6}{18}$. Right and left, cataracta cupuliforme. Right and left degenerative changes and separation of Berger's layer of the lens capsule.

3.10.31.—Right tension + 1; left tension normal. R.V. $\frac{6}{24}$, L.V. $\frac{6}{24}$. The right eye showed an extension of the lens opacity—the fundus imperfectly seen, but the disc not obviously cupped.

10.10.31.—R. 2 mm. corneo-scleral trephine; complete iridectomy.

The anterior chamber is not unduly shallow. In the coloboma there is a bluish-grey felt-like membrane on the anterior lens capsule, which here and there is peeling off the true lens capsule.

Left eye.—A similar condition of the lens capsule would be seen if the pupil were dilated.

Mr. GOULDEN said that the idea that there was a layer of the capsule not derived from the epithelium of the lens was suggested by the fact that cases had been reported of a blow on the eye which dislocated the lens, leaving the anterior capsule partly *in situ*, and the lens lying at the bottom of the vitreous chamber. But this lens did not become opaque, therefore obviously the whole thickness of the capsule had not been removed. Berger had suggested that the zonular fibres derived from the vitreous were attached to the lens by a sheet of capsule which extended a little on to the anterior surface of the lens about to the edge of the pupil, and in cases of irido-cyclitis with much infiltration there was a tendency for a stripping of this layer to take place by infiltration of round cells.

Chronic Glaucoma with Buphthalmos.—T. W. LETCHWORTH, F.R.C.S.

James G. R., aged 23.

January, 1929. First attended hospital. Tension.—Right, 70 mm.; left, 60 mm. Discs cupped and atrophic. Buphthalmos. Right field slightly constricted. Left field narrowed on nasal side to within a few degrees of fixation.

Right eye trephined February, 1929; left eye trephined February, 1929.

Tension rose again in each eye after the operation. Trephining repeated in right eye April, 1929, and January, 1931. Left eye trephined April, 1929 (below) and Lagrange in June, 1929.

Present condition.—Tension normal in each eye.

R.V. $\frac{-\ 1\ \text{sph.}}{-\ 8\ \text{cyl.}}$ 90° $\frac{6}{9}$ pt. L.V.: no central vision.

Large bleb over upper trephine in left eye.

Chronic Glaucoma.—T. W. LETCHWORTH, F.R.C.S.

Rose B., aged 25.

First seen March, 1926. Tension (each eye), 60 mm., Schiötz.

R.V. + 2 sph., $\frac{9}{36}$. L.V.: no perception of light.

Deep funnel-shaped cups with atrophy. Right field constricted all round to within a few degrees of macula.

Both eyes trephined, April, 1926.

Tension normal since then. R.V., $\frac{6}{12}$.

Mr. LETCHWORTH also showed the following cases: (1) **Coat's Disease**; (2) **Blue sclerotics and fragilitas ossium**; (3) **Parinaud's conjunctivitis**; (4) **Sympathetic uveitis**; (5) **Stargardt's disease.**

In describing the last case Mr. Letchworth said:—

The patient is a girl, aged 14, whose vision was down to $\frac{6}{36}$, but it was very difficult to detect anything abnormal in the fundus. Subsequently there was mottling, and later a development of pigment at the macula. I have known the condition in a brother and sister, and neither I nor my colleagues were able to find any change at the fovea in the early stages. In all the cases vision was reduced to between $\frac{6}{24}$ and $\frac{6}{18}$. The disease is familial.

Retinitis Pigmentosa with Hirschsprung's Disease.—L. H. SAVIN, F.R.C.S.

I am showing, by courtesy of Dr. Worster-Drought, a girl, aged 14, blind from birth, with retinitis pigmentosa, nystagmus, and left internal squint. The Wassermann reaction is negative, and there is no suggestion of syphilis in the family. A small brother also has retinitis pigmentosa. My reason for showing the case is that there is also congenital idiopathic dilatation of the colon. Periodically the abdomen becomes grossly distended and the temperature rises to 101° or 102° F. Since 1925 the patient has been obliged to have enemata to open her bowels. I suppose ultimately the gut will perforate or become obstructed, and cause death.

Retinitis pigmentosa seems occasionally to be associated with other congenital abnormalities such as deafness or supernumerary digits. I have not hitherto seen it associated with Hirschsprung's disease.

Right old Choroidal Atrophy with Left Choroido-retinitis juxta papillaris.—L. H. SAVIN, F.R.C.S.

Patient, a man aged 25, came to hospital two weeks ago with what seemed to be a typical retinitis juxtapapillaris in the left eye. The blurred area of white near the disc is extending rapidly. There is an atrophic area of choroid in the right eye, which by its lack of surrounding pigment suggests a tuberculous origin. He has been treated for supposed "enlarged hilum glands" in his youth. Wassermann reaction, negative. Urine, normal.

Reflex Iridoplegia with "Myotonic Reaction" of Pupils.—CHARLES YOW, M.D.

Patient, Miss E. R., aged 20. In February, 1931, a piece of grit entered her right eye and her friends noticed that the right pupil was larger than the left. This is a case of non-syphilitic Argyll-Robertson pupils. These pupils do not react to light, are semi-dilated but on accommodation give the prolonged slow contraction, and dilatation when this is relaxed, which Dr. Adie has provisionally labelled myotonic.

Anomalous features in this case are that the knee-jerks and ankle-jerks on one side are absent, and that syphilis can be definitely excluded from the history. The Wassermann reaction was negative.

Disciform Degeneration of Maculæ.—CHARLES YOW, M.D.

Mrs. E. C., aged 69. September, 1929: Left eye: patch of active exudation involving macula; vitreous opacities. Vision: counting fingers. Right eye, $\frac{6}{6}$. The right eye began to be involved about January, 1930. I saw her about October, 1930, when there was œdema of the right macula with small hæmorrhages surrounding it. R.V., $\frac{6}{60}$. In the left eye there was a raised greyish patch. About July, 1931, the hæmorrhage had cleared up in the right eye and there was a raised circumscribed greyish patch. In November, 1931, the left patch was becoming flatter. There was no change in the other parts of either fundus.

There is now a circumscribed greyish patch in either fundus. The retinal vessels appear to be normal. This case shows a typical history and picture of the condition.

Four Cases of Typical well-marked Disciform Degeneration of the Maculæ.—A. SORSBY, F.R.C.S.

The patients' ages are respectively 79, 77, 70, 70.

Different phases of this affection have been described under a mass of different names, such as choroido-retinitis with arterio-venous aneurysms, tumour-like proliferation at macula, senile changes, senile form of Coat's disease, senile exudative retinitis, retinitis with massive exudation, and even chondroma growing from elastic lamina and exudative retinitis with bone-formation.

P. Junius and H. Kuhnt ("Die Scheibenförmige Entartung der Netzhautmitte," Berlin, S. Karger, 1926), have stressed the unity of these cases. They are degenerative, and not inflammatory lesions; the macula is the site of election, though the surrounding area may be involved; the course is progressive, ending in typical cases in a disciform area of degeneration at the macula. In the incipient stages, only œdema may be present, or an œdematous area with yellowish glistening dots; sometimes there is a circular hæmorrhage at the macula, or crescentic hæmorrhages surrounded it. More rarely massive tumour-like proliferations at the macula, which have led to excision of the eye, are present. The six cases shown to-night illustrate the different stages of this affection. Judging by these and two more I have seen, the lesion seems to affect both eyes, though not necessarily simultaneously. In the very early stages so little may be amiss that at least one case reported in the literature had been diagnosed as retrobulbar neuritis. The end-stage involves complete destruction of the macula. Junius and Kuhnt regard the lesion as due to a disturbance in the blood-supply, leading to retinal degeneration with reactive tissue proliferation, even to cartilage and bone formation. Carl Behr, on the strength of a microscopic examination, holds that senile sclerosis of the lamina elastica cuts off the blood-supply to the macula; the subsequent changes are secondary. Localization to the temporal half of the retina and the age-incidence distinguish the lesion from Coat's disease. It would appear to be associated to some cases with retinitis circinata and angeoid streaks.

References.—JUNIUS, P., and KUHNT, H., loc. cit. DAVENPORT, R. C., *Trans. Ophthal. Soc. U.K.*, xlvi, 137, 1927. BEHR, C., *Zeitschr. f. Augenheilk.*, lxviii, 1, 1929.

Arachnodactyly.—ARNOLD SORSBY, F.R.C.S.

Female, aged 16. The build and the hands are typical of the condition, and there is subluxation of the lens in each eye. The pituitary fossa is normal.

Mr. A. W. ORMOND said it must be allowed that this was an example of the condition named, but it was much less marked than many of the cases—nearly 100—which had been recorded. One of the features which emerged notably was that it was a familial disease.

POSTSCRIPT.—Dr. Margaret H. Banks has kindly traced and examined this patient's immediate relatives. Neither the parents nor the brothers and sisters show any abnormality. The general condition of the patient herself is perfectly good. [A. S.]

Congenital Absence of all Four Puncta.—ARNOLD SORSBY, F.R.C.S.

Patient, a boy, aged 8. There is an interesting history of the father having had the same sort of thing, and also the grandmother. I can trace only one case in the literature, reported from this hospital by Brooksbank James in 1902. I do not know whether that patient is the father of the child who is shown to-day; I hope to report as to that later on. Probing has been attempted, but unsuccessfully.

POSTSCRIPT.—The father of this patient is not the case referred to by Brooksbank James. But he too has absence of all four puncta. The grandmother is dead; she had "watering eyes all her life." The patient is an only child. The father has one sister alive; she is normal. [A. S.]

Reference.—Trans. Ophth. Soc. U.K., 1902, xxii, 201.

Injury to Superior Oblique Muscle.—ARNOLD SORSBY, F.R.C.S.

Caught inner and upper angle of right orbit with the open blade of a pen-knife on November 6, 1931. Diplopia noted at once. The stab wound was quite a minor injury, healing within a few days. Diplopia has persisted.

Vertical diplopia most marked downwards. Separation most downwards and inwards.

It is a typical picture of injury to the superior oblique, either at the pulley, or the tendon near by.

Discussion.—Mr. HARRISON BUTLER said that he had had a case in which he had removed a cyst from the orbit. He was very careful to avoid the pulley of the oblique, but the cyst extended deeply into the orbit, and had to be cut off as far back as possible. The operation was followed by complete paralysis of the oblique. The cyst contained muscle fibres and apparently was growing from the belly of the muscle. No treatment was undertaken, and the case was lost sight of.

In another case the sufferer fell down in the street, and afterwards was found to have this paralysis. He did not know whether it was part of the original cerebral condition that led to the fall, or whether it was a direct traumatism due to the blow on the head. After a year there was still some diplopia on looking downwards.

Dr. E. E. MADDOX said he had had two or three cases of traumatic paralysis of the inferior oblique muscle, which was successfully treated by setting back the inferior rectus of the other eye. One case (not traumatic) was that of an ophthalmic surgeon, who went about with his head on one side. It was before the days of the recession operation, but he tenotomized the inferior rectus of the other eye, and this made the head straight permanently. In traumatic cases, if, after waiting a considerable time, any operation were decided upon, he would recess the inferior rectus of the other eye.

Mr. N. Bishop Harman showed an electrical adaptation of the ophthalmoscope model which he exhibited to the Ophthalmological Society of the United Kingdom in 1904. The unique feature of the model is the special mechanism whereby a lens range of 70 dioptres can be obtained in a machine of conveniently small size.

The following cases were also shown: (1) **? Coat's Disease.**—A. D. GRIFFITH, F.R.C.S.; (2) **Excision of Tarsal Plates for Trachoma.**—A. F. MACCALLAN, F.R.C.S.; (3 and 4) **Anomalous Reaction of Right Pupil, with Absence of Tendon Reflexes, in Boys aged 12 and 13 respectively.**—W. J. LINDSAY, M.D.; (5) **Ophthalmoplegia.**—W. J. LINDSAY, M.D.; (6) **Retinal Arteriosclerosis.**—E. A. DORRELL, F.R.C.S.; (7) **Foreign Bodies (Glass) in Left Eye.**—E. A. DORRELL, F.R.C.S.; (8) **Disciform Degeneration of Macula.**—L. H. SAVIN, F.R.C.S.

United Services Section.

President—Lt.-Colonel E. M. Cowell, D.S.O., R.A.M.C.(T.A.).

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Recent Research Work in Deep Sea Diving.

By A. E. Phillips, M.B. (Surg. Lieut.-Comm. R.N.).

The British Admiralty continues to place reliance in the rubber diving suit, and for the past three years, in collaboration with Messrs. Siebe, Gorman and Co., has been conducting research and practical experiments in deep sea diving, both with the rubber suit and with the German all-metal suit.

When one considers the objective of deep sea diving, which is equally the saving of life in a sunken submarine and the recovery of sunken trophy, it will be seen that the cumbersome all-metal suit has distinct disadvantages. Although allowing certain work to be carried out at a greater depth than can even now be obtained in a rubber suit, its comparative immobility and the difficulty in keeping its joints water-tight preclude its use in the rapid work necessary for saving lives entombed in a disabled submarine.

A similar dress is used by the Italians in their endeavour to recover bullion from the "Egypt," but it has no legs and is used purely as an observation chamber, the diver telephoning the instructions necessary for placing grabs and demolition charges.

Let us turn to the rubber diving suit, and the methods employed in the British Navy for diving between 200 and 300 feet, a depth where much useful salvage can be carried out.

As the diver descends he is subjected to increasing water-pressure, which is transferred to the air in his suit, and hence to the air in his lungs. This increased alveolar pressure causes a corresponding increase in the amount of the gases in the body. The pressure of abnormal amounts of nitrogen in the body causes no inconvenience as long as the gas is in solution, but should the pressure be released too rapidly for the gas to escape through the lungs, bubbling occurs. A bubble in its action resembles an embolus and is the cause of caisson disease—the bugbear of all who work under increased air pressure. An increase of oxygen or carbon dioxide would cause poisoning. The amount of oxygen necessary to produce oxygen poisoning is not completely defined, but experiments on animals are now in process.

Should the diver be brought too rapidly to the surface, bubbling of nitrogen will cause caisson disease. The prevention of this has been known since the days of Paul Bert. It suffices to reduce the pressure gradually by bringing the diver up slowly, this being known as decompression. The present Admiralty Tables were drawn up as a result of the work of Professor J. S. Haldane, Professor A. E. Boycott, and Captain G. C. C. Damant, R.N., for the Admiralty Committee of 1907, and are used all over the world with most successful results.

These tables were calculated for a maximum depth of 204 feet, and a maximum stay on the bottom of one hour, or in an emergency for one hour and fifty minutes.

Now the time taken in bringing to the surface a diver who has been working at a depth of 204 feet for one hour, is 124 minutes, implying a considerable waste of working time, and a considerable feat of endurance when it is considered that he is hanging on a rope suspended in mid water, often in winter, or even in our summer, at a temperature of 40°-50° F., and in a strong tideway. To overcome this difficulty, Mr. R. H. Davis, Managing Director of Siebe, Gorman & Co., has designed and produced a steel chamber which, when submerged acts as a diving bell, and when closed as a decompression chamber. The Davis Submersible Decompression Chamber is lowered to the depth at which it is required to pick up the diver. It is fitted with "Salvus" oxygen breathing apparatus, depth and pressure gauges, thermos flask containing hot coffee, etc. This chamber was designed: (1) for the greater safety and comfort of the diver; (2) to enable him to use oxygen breathing apparatus to accelerate decompression; (3) to work at greater depths for longer periods; and (4) to be brought up immediately and safely to the surface in emergency. Oxygen breathing permitted by this chamber shortens the time of decompression by a half to a third and materially adds to the possibility of successful diving at depths of over 200 ft.

The importance of the use of oxygen in decompression, originally advocated by Paul Bert, has been recognized by physiologists who have followed him, and confirmed by Sir Leonard Hill and others who have carried out research on the subject.

In the months preceding the 1931 diving trials, Mr. R. H. Davis arranged for Captain Damant to calculate a new set of decompression tables to a depth of 300 ft. on Professor J. S. Haldane's system, taking into consideration five groups of tissues saturating at different rates and allowing for the accelerating effect of oxygen breathed from a certain point in the decompression scales. Mr. Davis placed his experimental plant and other facilities at the disposal of the Admiralty. Several hundred tests of these tables were made on goats, and it was found necessary to increase the safety factors largely (the system of calculating remaining the same). Eventually a decompression table was produced which had been thoroughly tested over the range in which the men were to dive, and the success of the deep water trials described below was largely due to this careful preparatory work.

What knowledge have we gained by the work of the last few years?—(1) Deep diving has emerged from the chrysalis stage. Investigations are still proceeding and much remains to be done, but sufficient knowledge has been gained to permit preparations for the routine training of a proportion of our divers in deep sea work. It is hoped that later on deep sea diving sections will be attached to our principal fleets.

(2) Oxygen breathing during decompression and D.S.D.C. have made diving safer, and the time spent during decompression more comfortable. It has also effected a saving of from a third to a half of the time in decompression—a valuable gain now for the diver and the officer in charge of the salvage.

(3) Diving, and working at a depth of 300 ft. in a rubber suit, is both safe and practicable. One of our divers reached a depth of 344 ft. and came to the surface as one might return from an afternoon walk.

The use of oxygen in deep diving.—The intensive use of oxygen is viewed with mingled feeling in modern medicine, for while it confers many undeniable benefits, it is responsible under certain conditions, such as prolonged exposure, for undesirable poisonous effects.

In animals, exposure to 45 lb. pressure of oxygen quickly leads to convulsions, and as the pressure rises, the quicker is the onset and the more likely is pneumonia to supervene. With mice and rats the convulsion period is preceded by extensive washing operations, which after a time, depending on the pressure, change into

running convulsions. These last a few seconds and are usually succeeded by a period of inactivity When the pressure is reduced convulsions are again noticeable, often even if no previous convulsions have occurred.

A few minutes' exposure to a 100 lb. pressure of oxygen produces severe convulsions in the smaller animals.

Exposure to 45 lb. and under takes longer to produce toxic effects, mice convulsing in about 20 minutes. At the end of 30 minutes two rats had shown no symptoms, as was the case with two monkeys. 40-45 lb. seems to be near the critical pressure for animals; below this they are fairly safe, except that during long exposure they appear to go into a stupor; over 45 lb. they are much more liable to convulsions and pneumonia.

Luckily we have no cases of oxygen poisoning to record among the divers; even allowing for the use of oxygen during decompression we have a safe margin.

We have found in our limited experience with oxygen (1) that, like the small boy in the soap advertisement, the divers are happiest when they get it. (2) They state that after their dive, when they climb into the D.S.D.C. and commence breathing oxygen they feel very refreshed. This may in part account for the feeling of fitness on the bottom, since even though breathing atmosphere air, they are even then exposed to two atmospheres of oxygen. (3) The exposure of one diver to 88 minutes' breathing of oxygen at an average pressure of two atmospheres, produced no evil effects, except subsequent sleepiness. (4) After three months' diving there was a slight diminution of lung fibroses in all ten divers. (5) In one case functional albuminuria was prevented by the use of oxygen.

What standard of fitness has been regarded as essential for deep sea diving?—Except that the diver should be possessed of a very stable mentality our experiences do not suggest that it is necessary for the deep sea diver to be any more fit than a shallow water diver.

Volunteers for deep diving must have had over two years' experience in shallow diving, and be under the age of 30. While deep diving was in the experimental stage, to be on the safe side, only the very fittest were chosen. This was partly to ensure that if the work proved very arduous, the diver would have the stamina to stand up to it, and partly to eliminate all those subject to illnesses which, if they occurred during the trials, would hang up the man's diving. From the information gained it is hoped to be able to reduce the standard of fitness now that routine training has been instituted. Previously the thin spare type was regarded in our Navy as the most suitable for diving. Our experiences suggest that the thin type has not always the same physiological and psychological reserves. Deep diving, since the pressure against which the diver has to work is greater, requires a more powerful build of man. Personally I like to see a moderate supply of adipose tissue; the work is sufficiently hard to work off any excess, and a small fatty layer protects against the cold. The entrance medical examination was mainly directed to obtain a stable heart, a sound labyrinthine system, and healthy lungs. It was required that these systems should stand up to some hard work and, if necessary, hard knocks.

The examination was similar to that of the Air Ministry, and my thanks are due to them for their assistance, and especially to Wing-Commander Tredgold.

Some of the candidates accepted had one defect, but this was only permitted if it was not considered likely to produce an adverse effect on the diver. One of the divers had a well-marked hyperpiesis but he came through the season just as well as the others, and actually was much healthier afterwards than before. His blood-pressure at the beginning of the season was 143/93, and at the end of the season 132/80.

A test that I call the 250 test, which I found useful in inducing albuminuria if any disposition to it existed, revealed an interesting condition in one diver. The test

consisted of touching the toes with the fingers 250 times in ten minutes; to be satisfactory, the pulse-rate should not exceed 130 on completion, and should return to within 10 per cent. of the pre-test rate in twenty minutes. One diver was found after this test to have his urine loaded with albumin. When this test was repeated and oxygen was breathed instead of air, no albumin could be detected. This test was carried out by him thirty-seven times in six months and the result was always the same. Renal efficiency tests suggested that this was a case of functional albuminuria. Other cases of this complaint were subjected to similar tests, and in every case the amount of albumin was diminished or eliminated altogether by the use of oxygen.

Another test employed, which is not in general use, was the mental excitability test; it was a modification of a similar test invented by the Japanese. Small strips of blotting paper, treated with carbol fuchsin, and mounted on plaster were placed on the palm of the hand, and a control strip was placed on the chest near the axilla. The test relies on the assumption that sweating of the palms is due to mental excitement or pain and is not due to heat. I considered the result positive if the stain on the palm was about twice as pronounced as that made by the control strip. This test gives moderately accurate results.

Value of certain medical tests and results obtained.—These trials have afforded an opportunity for exhaustive and continuous application of certain tests; moreover they supplied what must be regarded as something approaching the optimum figures for the tests, for owing to the rigorous medical excluding examination and the healthy lives they lead, it would be difficult to find a healthier body of men.

At the initial medical examination very many tests were applied, not so much to exclude candidates as to throw light on the efficiency of the test for separating the likely successful divers from the likely unsuccessful divers. Therefore, blood-pressures, pulse-response tests, 40 millimetres, balancing and labyrinthine tests, and vital capacity tests were applied daily, before and immediately after diving. As would be expected, daily variations were always present and existed during different times of the same day, but they were slight. Towards the end of the diving week, blood-pressures were on the up-grade, and vital capacities falling, all very slightly. The long week-ends effectually restored the levels to par. Further this tendency could be observed at the end of the three months' diving season. This was most noticeable in the diastolic blood-pressures. Incidentally the men with a slight hyperpiesis showed the better stamina.

I attach great importance to a stable labyrinthine system, since the diver may have to work in bad lighting conditions. Contrary to the usual experience, the holder of the best records for the 40 millimetre test showed the least desire to hold out under unfavourable conditions. To hold with one's breath a column of mercury at 40 mm. for 2 mins. 23 sec. is almost unheard of. This diver exhibited wonderful endurance in achieving this figure, but his endurance on the bottom was strictly limited; psychologically he was not so stable, and it might have been something in this direction which hampered him when diving. He had repeated attacks of caisson disease and therefore was removed from the deep diving list. Very little difference could be detected in the figures obtained before and after diving. One noticeable after-effect, probably due to oxygen, was an overwhelming desire for a nap, a few hours afterwards, even after sitting in the experimental chamber in London.

Working conditions on the sea bottom at 300 *ft.*—At 300 ft. the divers have to work against a pressure of ten atmospheres, and in addition have to contend with the low temperature of the water; so it is desirable that they should have some protecting layer of fat.

By means of a toy which I have had made, I have studied the heart condition of the diver when working and when at rest on the sea bottom. It consists of a microphone fixed over the apex of the heart, connected through the diver's

helmet to an amplifying set on the surface, and hence to a loud speaker or headphones. By means of this device I found that the diver at work on the bottom had, in spite of the pressure of ten atmospheres, an increasing pulse-rate of only five beats per minute over his pulse-rate for similar work on the surface. I attribute this to the slowing influence of oxygen, for at this depth the diver has the benefit of the equivalent of two atmospheres of oxygen—the oxygen in the air being responsible for one-fifth of the total air pressure.

I am having another device made to record the respiratory rate of the diver. It consists of an electric belt worn round the thorax, and connected through to the surface to four electric bulbs. It is so arranged that at rest, bulb 1 lights on inspiration and dims on expiration, under working conditions bulbs 2 and 3 are illuminated depending on the depth of inspiration, bulb 4 means maximum expansion.

The condition of a diver on returning from work at 300 *ft.*—He is as fit as and often fitter than when he went down. Diver after diver has told me the same, and their statements are substantiated by a number of records. I quote a case at random: Blood-pressure before diving, 123/79; after, 116 and 85. 40 mm. test before diving, column maintained for 62 seconds, after dive 64 seconds. Pulse-rate per 5 seconds, 7, 6, 7, 6, 7, 6, 8; after dive, 6, 6, 6, 6, 6, 7. Pulse-rate resting, before dive 74; after dive, 72. Pulse response test: pulse on completion of test, 116; at end of one minute, 92; after diving, 108 and 82. Vital capacity before dive, 5,250; after dive, the same.

Are there any dangers peculiar to deep diving?—None that we have yet encountered. More care is necessary to prevent the diver coming to the surface too rapidly, since he is more heavily charged with nitrogen. The danger zone for oxygen poisoning is more nearly approached, but we are careful to keep outside the zone. So far no case has been encountered and we do not anticipate having to cope with this danger. Carbon dioxide poisoning requires attention, since 1 % in the helmet on the surface becomes 10 % at a depth of 300 ft. There was one mild case this year.

Slight attacks of giddiness may be experienced on the bottom. This we believe in most cases—in one we have positive proof—to be due to the diver descending too rapidly, and, by putting too great a strain on his tympanum, upseting the labyrinthine system, reflexly.

In 1930 one diver died from complications following caisson disease, and by some it was considered that psychological forces presently to be described played a contributory part. With regard to this case I wish to make it clear that the caisson disease encountered in deep diving in no way, as far as we have encountered it, differs from the forms manifested in shallower diving or tunnel work. It cannot be too strongly stressed that the cure for caisson disease, whether caused by tunnelling, shallow, or deep diving, is the same—immediate and adequate recompression. No matter how serious the case, if recompression is properly carried out, the treatment should succeed.

Caisson disease may assume a host of forms. If the reader considers the different places in which the bubbles may form, he can evolve the symptoms which will ensue from the mechanical obstruction caused thereby. Some places are, however, more prone to caisson disease than others—for example the blood, muscle, nerves and organs may be affected. Bubbles tend to form where circulation is poor, but except where decompression is seriously inadequate, the blood and viscera escape. Even where decompression is at fault, recompression will save the case, especially is this so where the D.S.D.C. is used, for the diver is under observation and treatment is greatly facilitated. This year in deep diving we had 16 cases of "bends"—an extremely painful manifestation of caisson disease caused by a bubble in a sensory nerve or nerve ending. These cases were all cured by a recompression pressure of a few pounds. Of these, five occurred in the same diver and this necessitated precluding

him from further deep diving. It seemed to me that these bends were more likely to occur on damp days. It is certain that when divers suffer from a neuritis-like after-effect, these attacks always coincided with wet weather.

Oxygen breathing was carried out during recompression, and here, as in decompression, an appreciable saving of time is effected. The recompression chamber is comfortable, and the diver has one or two attendants and can read or play cards during the lengthy process of recompression.

Some men appear to be more liable to caisson disease than others; it is one of the dangers to which all divers and tunnel workers are subjected if adequate decompression is not given, and it is not peculiar to deep sea work, nor was it caused by the new train of symptoms which I am about to describe. However it is considered by some that these symptoms played a contributory part in the case of the diver who died.

The sequence of these new events and their influence on diving were as follows: During the 1930 season, when working, or in many instances resting, at from 270 to 300 feet, the diver experienced what to him were new sensations; he found that it was much more difficult to assimilate facts and to exercise the quick decision essential for successful diving. It might be summed up as a slowing cerebration. Some of the divers went a stage further, for when they returned to the surface they stated that they had "passed out" when on the bottom. It was known that if this was so it could not have been for long, for they had answered by their telephone the instructions they were continually receiving. Others stated that they had experienced a detached feeling, as if they were under an anæsthetic. Another when asked to describe deep sea diving said:—

"You notice the dark more, though it may not be darker. The light is a comfort and company. You notice things more if there is nothing to do, I get comfort from seeing the fish, it takes your mind off everything else."

This diver also had patchy loss of memory, his main statement is more a history of mental tension, but it is valuable inasmuch as it shows that in 1930 tension existed in some of the divers at any rate, whereas in 1931 we failed to get any evidence of its existence. Another diver said: "You get keyed up in deep water," he also had some difficulty in remembering the work he had done.

An old hand at diving, when asked for a description, gave the following account:—

"You have to be more careful in deep water; in deep water you know that you are concentrating." He described how "you think of each heave as you turn a spanner." Adding . . . "If you go down with a set purpose it becomes an obsession, it will become the main thing and you will forget everything else."

He described how he thought very deliberately:—

"I have finished my job, what shall I do next?—of course I have finished and now I must go up."

He described how he was aware of every action.

"If my hand goes out I think of my hand going out."

He gave the following as an analogy:—

"If I saw a thing of value, say half crown, in the street, I would pick it up. Down below I would look at it and think—'What is that, shall I pick it up?—yes, I will pick it up,' and then I would feel my hand go out."

The latter is, I think, the best description of how most of the divers felt in 1930 when between 270 to 300 feet. Some felt it less, others more. With two exceptions

all the divers looked and felt fit when they returned to the surface. The exceptions were white faced and "windy" when they came out of the D.S.D.C.; these two were regarded as unsuitable for further deep diving.

These accounts given by the divers had to be sifted and action taken to discover the cause of their loss of memory. Sir Leonard Hill was of the opinion that the cause was mental and not physical. The Admiralty Deep Diving Committee asked for and obtained the assistance of the Medical Research Council in investigating these new disorders, and Professor Culpin was appointed to investigate the problem. From the descriptions given him he considered that these symptoms were more likely to be expressions of a mental than a physical disorder, and he stated that he had met with a similar condition of so-called loss of consciousness in cases of shell-shock. Doctor Culpin then interviewed these divers and reported that with three exceptions they were free from symptoms. Two of the three were the men who, as a result of their experience on the bottom, had been declared unfit for further deep diving. In these two cases Professor Culpin succeeded in restoring missing pieces of memory. The third exception had only had a very slight loss of memory. The same method which had succeeded with the other two men was employed, but the endeavour to piece together his memory failed. This man was permitted to continue diving; this year he felt no abnormality and was regarded as our most successful diver.

The following is a complete report of a diver who arrived at the surface blanched and windy, and was excluded from further deep diving. He was one of the divers whom Professor Culpin successfully treated for loss of memory. I am indebted to Dr. Culpin for this summary of the account given at his interview with the diver.

The interview was directed to obtain an insight into the psychology of the diver as well as to restore, if possible, missing links of memory. The detailed account which the diver gave when in a mildly hypnoidal condition, warranted the assumption that his memory had been restored. Regarding his own psychology, the diver volunteered the following information:—

"I don't like to attract attention, nor would I care to go alone into a teashop which I did not know, for I would feel that everyone was watching me; I would rather go hungry! Discipline irks me, I am afraid of doing the wrong thing, I often have the feeling of being watched, it affects me when in charge of strange men. I keep to myself, and I am afraid, sometimes worry what others think about me."

As a child he dreaded his father. Coming nearer to the events which affected his diving, he said:—

"I never remember being afraid of the dark, but I have always been afraid of enclosed spaces; I get a feeling of being sealed in."

The terror (his own words) came on first when skylarking with others he found himself at the bottom of a scrum and was nearly suffocated. Once as a child he was thrown into the sea, and since then has not liked it, and although he has passed swimming tests he does not like going out of his depth. As a diver he was frightened of making mistakes. The old fear of being closed in came back to him:—

"It had not worried me for a while, but it came on that time just before I went off—on the bottom—that stirred it up, and I have had it ever since."

He then described his deep diving in 1930.

"I felt dizzy at 240 ft., and at 270 ft. I felt like being in a nightmare. It felt like going under ether, I think that is what made me think of going unconscious. I had a feeling of being closed in and went off. I did not tell them what I told you, I just said that I had been unconscious. On that occasion I felt tingling in my limbs, and I thought that the heavy pressure was crushing me into my suit. I thought that if I ever get out of this I'll never dive again. After this I cried off deep diving, but thought it over and afterwards asked to be allowed to carry on."

He described his last dive, which was at 300 feet, as follows :—

" I left the ladder determined to get to the bottom; at 250 ft. I got a recurrence of the tingling, and a feeling of lying on my back. I decided to rest for a couple of minutes and then go on. I slid 10 ft., and felt I was going unconscious. I made signals to be pulled up and kept repeating them, I lost the use of my limbs and let go everything. While hanging on the shot rope, I saw my own face in the front glass, it was outside the glass and looked all greenish. I was dressed in my shore-going suit! I heard the order, 'Pull the diver up,' again and again, as if someone in the suit were saying it. When I got to the submerged chamber I did not appreciate the oxygen as usual, I wanted fresh air."

At the interview with Professor Culpin he was placed on a couch, with closed eyes, and directed to go over the descent " as if it were happening now." With some urging he repeated the performance, and seemed to recall the whole of the period for which he had claimed to be unconscious. At one stage he cried :—

" Pull me up, for God's sake pull me up. I feel as if I'll never get up. I'm tied to the bottom, my mouthpiece is caught under my nose. It is getting lighter now, I can see the chamber."

Then he was made to sit up and tell the story again. This time he gazed straight ahead and talked as if he was still going through his experience, and he was able to add a few details to his first account :—

" I felt that I was being pulled up against resistance, as if a fellow was trying to hold me down, I am fed up and want to get out—worried because I can't open my by-pass; I seem to take two minutes trying to open it.'

In the talk which followed he agreed that incidents he had forgotten had now come back to him. It was decided that neither this diver nor the other who had also been under observation, was sufficiently stable mentally for further deep diving.

Candidates found to have a similar psychology are to be debarred, at least for the present, from deep diving. To prevent this type being selected, an interview, designed to look for these symptoms, was included in the medical examination for deep sea divers at H.M.S. " Excellent," and those passed by me were further vetted by Professor Culpin.

The ten divers for the 1931 season were examined and passed as free from symptoms, although the tenth was only passed after consultation between Professor Culpin and myself. The first nine divers encountered nothing abnormal, and no loss of memory or other unusual sensation was regarded during this diving season. Most of them, however, when in the experimental chamber at Siebe, Gormans, at an air pressure equivalent to a depth of 300 ft., did experience a momentary giddiness. If they were reading the print became blurred for an instant. This sensation was not repeated when working at 300 ft. After seven weeks' diving the tenth diver developed acute claustrophobia when at a depth of 270 ft. He is of the suppressed nervous type, who habitually exercises self-control. This attribute he has developed to a remarkable degree. The other divers told me that he was inclined to be erratic, and that he much disliked being the first to go down to an increased depth, or to be the first diver of the day. On the day when he broke down he unfortunately was the first diver of the day. He was only partially conscious of his own nervousness, for instance, he does not like going into the officers' mess; if he is in uniform ashore he feels he is being watched; more important—he was frightened of the dark as a child, and even now is very frightened of horses, nothing could induce him to pat a horse. In reply to the questions as to certain of his mental speculations he admitted, " I often think of where I come from, but I must not *talk* of it or they would think I am qualifying for an asylum." A history such as this seems to point to mental instability, but apart from these peculiarities—and they are, or certain of them are

possessed by many of us—he appeared to have a very equable temperament, and the deciding factor in accepting him, was his splendid physique.

During the chamber tests he gave a strong positive reaction to the Mental Excitability Test, but he insisted that he had not felt anything unusual. After the present breakdown he confessed that he had felt queer on one occasion. When asked if the feeling had been similar to that immediately preceding his breakdown, he said :—

"You cannot possibly compare the two conditions: in London, in the chamber, it was light, and there were others with me; on the bottom it is dark and lonely."

While he was diving I kept him under as close observation as I could without rousing suspicion. On one occasion his pulse-rate on the bottom was much too fast for the work he was doing, and I suspected from his conversation on the telephone, which sounded very artificial, that he was not altogether happy. I considered removing him from the trials, but this was a drastic procedure, especially as my suspicions were based on such flimsy evidence. On his last dive he had been six minutes at the bottom at 270 ft. when he urgently demanded to be brought up, he gave no reason but constantly repeated his demand. He did not appear to be in a panic but was most imperative in his request. Right up to this point he had been conversing on the telephone, and giving instructions to the surface regarding the hoisting or lowering of wires connected with his work. We had great difficulty in making him remain at his decompression stops, his one desire was to get up and get to the submerged chamber, if not to the upper deck. The chamber was specially lowered to a greater depth than usual, to comply with his request. The chamber attendant reported that, on arrival in the chamber he looked normal, but very white, and his eyes looked glassy. During decompression he became more cheerful and tried to describe what had happened on the bottom. He said,

"Have you ever felt you would like to murder a 'so-and-so'? Well that is what I felt like on the bottom when I came to, and found myself trying to unscrew my front glass; my one idea was to get out of the helmet and into the chamber."

When he emerged from the D.S.D.C. he seemed like one who has sustained a severe mental shock; his ocular appearance and whitened face supported this impression. Physically he was badly shaken, but no more. He was overcome by the situation, and deeply self-conscious of failure; indeed the main difficulty in restoring him was to overcome this idea. He was striving desperately to recover his self-control. He could not bring himself to recount his experiences verbally, but agreed to write them down.

The following extracts are from his own written statement.

"I was at the time kneeling on my right knee and head down (the required position for putting clips on the door) when suddenly I came over rather funny. It was a distinctly different feeling. I stood up, the tank wire in my right hand, and thinking it was a touch of CO_2, I began to breathe deep and hearty, thinking of course that in a couple of minutes I would be able to resume work. Then I seemed to go quite limp, a feeling of no life or energy. This was new to me, whether it was a part of CO_2, I didn't know, because I have never experienced a real dose of CO_2; anyhow, after stopping and doing the drill for CO_2, I thought I would be alright, but suddenly something seemed to—say—snap inside my head, and I started to, what I thought, go mad at things.

"I had small laps of this, on and off. Breathing became difficult, possibly I might have asked for more air, I couldn't say for certain. I really did try, and fought hard to beat off this madness, but it all seemed of no avail. I didn't get worse, but such as it was, it was quite enough for me. I wasn't in a real panic, and ready to do anything that came to hand, although I did make a hash in some things. Anyhow my one ambition at that moment was to get my helmet off, the quicker the better. I fought hard to stave off this feeling, but it wouldn't go. I should say that unless one had experience of this kind of thing it would be very difficult to imagine and realize such. I felt slightly relieved when, after closing my

ejector, I left the bottom. After going say—30-40 ft. up, I came to the conclusion that I was coming up a wire. I stopped, and I was pretty well O.K. I thought to myself, why I should think so I suppose was for the simple reason that the shot rope is the shot rope and every diver knows what it is, and that I was going all wrong.

"Naturally in that moment of, say recollection, I decided, although much against my inner feelings, to go down the wire, and leave the bottom a bit like a diver is expected to. Down the wire I went, and arrived on top of the tank, slid off, and stood up. I don't remember how I actually left the wire, and got to the shot rope, anyway, I must have found the shot rope because I came up it. At this time, i.e., when I was standing at the foot of the shot rope ready to ascend, I was perfectly normal, I felt my ejector, was it already closed? Or did I close it then? I can't remember, at any rate I did ensure it was closed before I left the bottom.

"I left the bottom, and as regards my ascent, can remember travelling light, or I should say light at one period. Of course, as regards the phone, which the Petty Officer attends to, I was simply saying things that I wanted to, and was not interested in the answers, I say not interested, but to take everything into consideration, I didn't look or wait for any answers, one must say that I was pure and simply giving orders. I had a check at — ft. I don't know, but at the time I dimly remember 110 ft., of course that being the check that coincided with the decompression tables. Since then I have been told it was 90 ft.

"I felt something happen on the shot rope, and I guessed it was the chamber being lowered to a depth that would be of some help to me, which afterwards proved to be correct. I received the check signal at—90 ft., and although I answered it, I really, inwardly, didn't want to. Once again my sane ambition was to get either on deck, or in the chamber, and have the helmet and glass taken off. Of course now at the present moment I am alright, and as one might guess, I feel a wee bit self-conscious of myself, but still at the time, I felt that I never wanted to be dressed as a diver again. Between now and then my views might change, but that remains a future answer.

"After doing a check which was very short, presumably a minute, again I had the greatest of all fights to stop there and do it. I had the phone message to go to the chamber. Just above me was the chamber, and I gladly got on to the ladder, and although in such a 'paddy' and 'panic' with myself, I did try and do things as I always had done. I undone the front weight lanyard, and let the attendant take the weight off, then I got secure in the chamber and got my front glass off. After stepping on the ladder and letting him take my front glass off, I didn't feel as happy as I thought I would, because things seemed in the same condition somewhat. It was a relief, however, to have the front glass off, and the helmet soon followed. While on the bottom I thought it would be absolutely good to have the helmet off, but, when it was off I didn't feel as I have before. I told the attendant everything was O.K., because physically I was alright, and again, my sense of self-consciousness came into play. But taking it all round, I just didn't have and couldn't display the usual amount of life that I have done on previous occasions. From then onwards I felt pretty well alright."

When he arrived on the upper deck he was on the verge of, at least, a complete mental breakdown. To keep his mind temporarily off the subject, he was sent on shore with two of his fellow divers and plied to the brim with alcohol. The danger in his case was that he would attempt to suppress the incident and relegate it to his subconscious mind. To avoid this I persuaded him that night verbally to recount his experiences on the bottom. Practically no hypnoidal effort was required to produce the horror of that morning's dive, and the picture of stark mad terror which even the interview could produce, left an impression which is very difficult for me to describe to you. My impression was of sitting in the stalls and watching the acting of a Grand Guignol. To such a pitch did he arouse his emotions, that he clawed at his face to remove the imaginary face glass and tore his clothes which he mistook for his diving suit.

The production of an abreaction produces as satisfactory a result as the surgeon's knife in abscess formation, and it was so in this case; from this on, the dam was loosened, and he was enabled to talk to the others of his experiences; previously he had refused to talk on the subject.

Since the incident two shallow water divers were reported as " windy." In each case I found the cause to be claustrophobia. In one, who was very particular about his air supply, I found that as a boy he had nearly been suffocated in a pillow fight, and ever since had been terrified about not getting enough air.

To sum up: The four failures in deep diving, and two in shallow diving, have many points in common. These men were of the suppressed nervous types, who habitually exercise control. Shy, reticent and self-contained, they work best by themselves and do not relish observation. They are usually of a philosophic, rather than a practical, disposition.

The most desirable method of selection to avoid mental instability, is a matter of opinion, but my experience suggests that for the present such a state of mental instability, slight as it is, debars from deep diving. Some advocate a very complete psycho-analysis, others preferring to rely on a very complete history of the diver from his first dive, coupled with a very close observation by instructors and officers, such observation being minutely recorded. The latter method depends on skilled and accurate observations which must be carefully recorded, but when the observer and observations are known, this is the method *par excellence*. At the time of these experiments this method of collecting facts had not yet been instituted, hence resort was made to the former method, which depends on an accurate forecast, by an expert, after careful study as to how the candidate is likely to react in given circumstances, mainly of the darkness and loneliness which may be encountered on the bottom of the sea, both of which are now largely mitigated by powerful arc lamps, an efficient telephone and observation chambers.

I do not wish you to think that the problem presented by these factors is to be regarded as comparable to the prominence which I have given to it in this paper. I have spent extra time on the subject because it is new. It is not peculiar to deep sea work, for we have found it in shallow water divers. It is something already in the constitution, which comes to the surface; fear of the unknown would be just as likely to produce the same symptoms in the persons I have mentioned.

I would like to acknowledge my indebtedness to Sir Leonard Hill, Professor Culpin, and Mr. R. H. Davis, Managing Director of Siebe, Gorman & Co., Ltd., for their assistance in the preparation of this paper.

Section for the Study of Disease in Children.

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The Plasma Phosphatase in Rickets and Scurvy.

By JEAN SMITH, M.D., and MONTAGUE MAIZELS, M.D.

THE distinction of having first described an enzyme which is intimately concerned with calcification and the growth of bone must be accorded to Robison [8], of the Lister Institute, who in 1923 pointed out that the cause of deposition of calcium phosphate in growing bone and its non-deposition elsewhere had up to that date not been explained, although as long ago as 1908 Adler [1] had calculated that an aqueous solution containing the same amounts of inorganic salts as are found in serum would deposit a mixture of calcium phosphate and carbonate.

Previous to Robison's work, the two explanations for the deposition of calcium in growing bone were as follows:—

(1) An increased solubility of the salts in the presence of serum (Pauli and Samec [6] 1909), and (2) specific adsorption of ions by the cartilage (Pfaundler [7] 1904).

Robison's hypothesis may be stated briefly as follows:—

The osteoblasts and hypertrophic cartilage cells secrete an active enzyme, a phosphatase, which by hydrolysing the phosphoric esters of the blood brings about a local increase in the concentration of the phosphate ions. The solubility product of tertiary calcium phosphate is thereby exceeded and deposition of this salt occurs in the ossifying zone.

This enzyme is present in ossifying cartilage in considerable quantity, and to an equal extent in the teeth [9]. It is present in greatest amount in bone at the time when the deposition of bone is most active, and is absent from cartilage which does not calcify, e.g., the tracheal cartilage (Kay).

Phosphatases are also found in the intestinal mucosa (Hunter [2]), and the kidney. The latter contains about 50% of the amount present in ossifying cartilage (Robison).

The phosphatases in the intestinal mucosa probably play an important part in the hydrolysis of the phosphoric esters of the food, and those of the kidney in the excretion of inorganic phosphate (Hunter). Robison's hypothesis has received further confirmation from the result of his experiments on calcification in vitro [10]. He was able to demonstrate that a rachitic bone split lengthwise and immersed for a short time in a solution of calcium glycero-phosphate or calcium hexosemono-phosphate, showed a deposition of calcium phosphate in the zones of preparatory calcification and of hypertrophic cartilage cells and in the periosteum (Robison and Soames).

Applying Robison's experimental work to clinical medicine, Kay [4] and others have shown that in certain generalized diseases of bone, the plasma phosphatase, an enzyme having all the properties of the bone enzyme and apparently identical with it, is markedly increased. The conditions which were first found to be associated with an increased plasma phosphatase were osteitis fibrosa, osteitis deformans, osteogenesis imperfecta, osteomalacia and rickets.

In view of Kay's paper in the *Biochemical Journal* in November, 1930, it occurred to us that it would be instructive to take a series of children with rickets and trace the course of the phosphatase content of the plasma and its relation to the healing process. Later, the study was extended to other conditions of which scurvy gave the most interesting results.

Method.—The short time at our disposal does not allow of a full description of the method used, and it must suffice to state that the one we adopted was that described by Kay [5], but we found it necessary to adapt it for use with small amounts of plasma. The enzyme activity is expressed in terms of the number of milligrams of phosphorus liberated as inorganic phosphate from excess sodium β-glycerophosphate in forty-eight hours at 37° C. by 1 c.c. of plasma at pH 7·4, corresponding to a pH of 7·6 at room temperature.

FIG. 1.—C. C. Rickets, 26.1.31. Active rickets, epiphyseal cupping, fracture of ulna, decalcification. Phosphatase 0·57.

Normal figures.—Kay [3] gives 0·15 mgm. as the average value for normal adult plasma, the range varying between a minimum of 0·1 mgm. and 0·21 mgm. In young infants the figure is higher. We found that the average value for six infants whose ages ranged between 3 weeks and 10 months was 0·18 mgm., while that of fourteen children aged between 9 months and 2½ years was 0·24 mgm. The following notes have been chosen from those of a series examined as illustrating the reaction of plasma phosphatase in rickets and scurvy to treatment. The first two are cases of uncomplicated rickets, selected from a series of twelve cases examined.

(I) C. C., aged 5 months. (1) First skiagram January 26, 1931, showed marked decalcification of the bones with irregular saucer-shaped epiphyseal lines and fractures of tibia, fibula and ulna (fig. 1). The blood at this time showed a very low calcium and phosphorus content, whereas the phosphatase was high (Ca 6·2, P 2·05, phosphatase 0·57).

(2) On March 17 there was good progress in healing, the fractures being scarcely visible in the skiagrams. The calcium and phosphorus were both almost normal and the phosphatase lower (0·44).

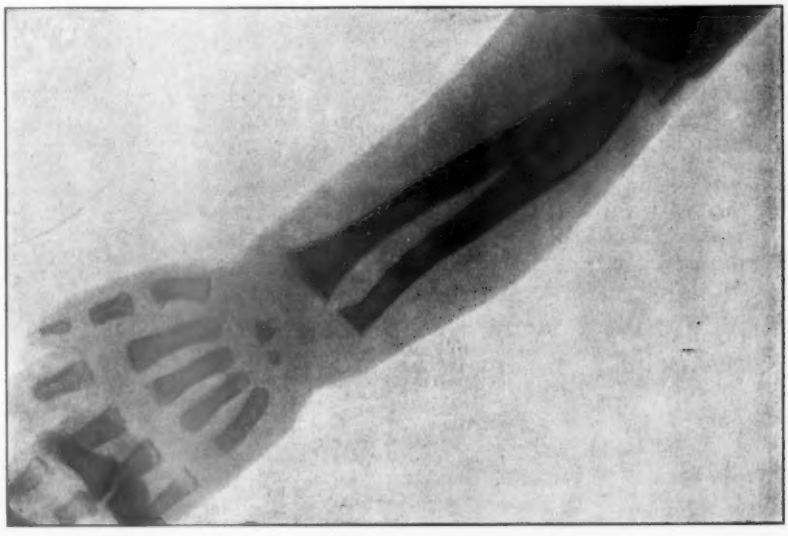
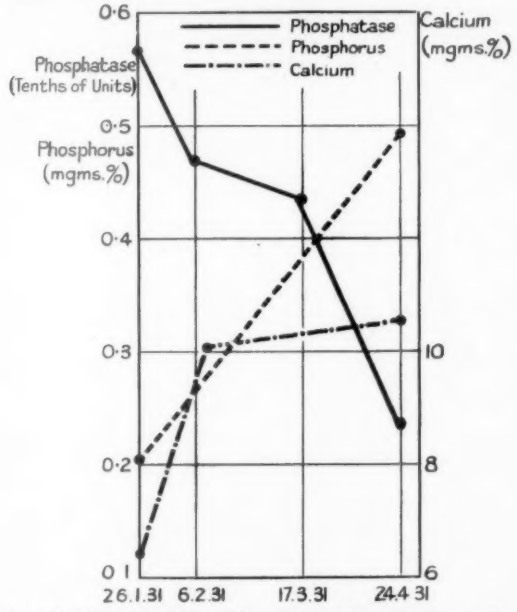


FIG. 2.—C. C., 24.4.31. Rickets healed. Phosphatase 0·24.

(3) On April 24 the rickets had healed (fig. 2) and the calcium, phosphorus and phosphatase had all reached the normal level.



C. C. Rickets. Chart showing changes in serum calcium and plasma phosphorus and phosphatase during the course of healing rickets.

(II) P. C., aged 1 year and 4 months. (1) On February 18, 1931, there was epiphyseal cupping, expansion and rarefaction of the bones. The blood calcium and phosphorus were both lower than normal (8·4 mgm. and 3·4 mgm. respectively) and the phosphatase high, 0·41.

(2) On April 14 the bone showed rapid progress in healing and the blood changes were equally marked; both the calcium and phosphorus had returned to the normal and the phosphatase was decreasing.

(3) On May 5 skiagrams showed the healed rickets but the phosphatase was still slightly above normal. This tendency to remain slightly above normal after the X-rays had shown that the rickets was healed was noted in some other cases.

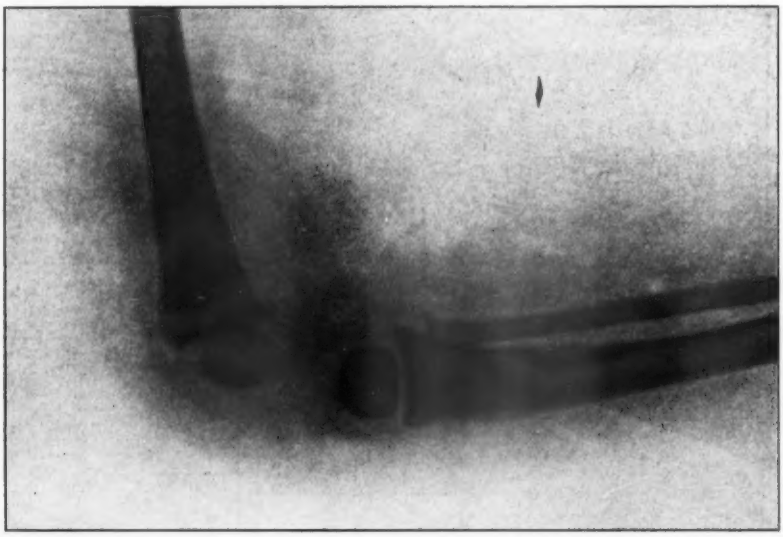


FIG. 3.—A. L. Scurvy, 26.6.31. Large hæmorrhage at lower end of right femur diagnosed clinically although not discernible at this date in the skiagram, which does, however, show the other characteristic radiological signs of scurvy. Plasma phosphatase 0·12.

Scurvy.—We have been able to examine five cases of uncomplicated scurvy. In those cases in which only minor hæmorrhages, e.g., spongy gums or petechial hæmorrhages in the palate, were present, the diagnosis was made by the combination of clinical signs—such as tenderness of the limbs, anæmia, and the history of a scorbutic diet—with the characteristic radiological appearances.

Two had massive subperiosteal hæmorrhages, in one case bilateral, in the other unilateral. Four of these cases, when examined in the acute stage, were found to have an average phosphatase value definitely on the low side of normal. The sixth was examined in the subacute stage and the phosphatase was normal.

The two cases with subperiosteal hæmorrhage were extremely interesting from the blood chemistry point of view. Both cases showed a normal figure during the acute hæmorrhagic stage with a sharp rise in the blood phosphatase when calcification set in.

(I) A. L., aged 11 months. (1) Admitted June 23, with a large subperiosteal hæmorrhage of the right femur. The radiological changes were typical of scurvy—Fränkel's "white line"

at the ends of the bones, the outlining of the epiphyses, the "streamers" at the ends of the femur and expansion of the costo-chondral junctions (fig. 3). The phosphatase at this stage was definitely low (0·12).

(2) On July 15 a large calcifying mass was seen to be ensheathing the lower end of the femur and extending almost as far as the trochanter on the outer side (fig. 4). The phosphatase was rising.

(3) On August 17 the calcified swelling was much more sharply defined, the edges were less rough, and definite laminæ were visible in the mass itself. The phosphatase had now risen to 0·47.

(4) On September 27 the mass was much smaller and the phosphatase showed a definite fall to 0·27.

FIG. 4.—A. L. Scurvy, 17.8.31. Large calcified mass at lower end of right femur. Phosphatase 0·47.

(II) V. C., aged 9 months. This case showed a similar sequence of events.

(1) Admitted September 28, with typical acute scurvy, the skiagrams showing dark hæmorrhagic areas. The phosphatase was low, 0·15.

(2) On October 16 there were large calcifying hæmorrhages surrounding both femora and extending along the outer side as far as the trochanter. The upper end of one tibia was also involved. The phosphatase had now risen sharply to 0·52.

(3) On October 26 the swellings were more sharply defined and there was a corresponding fall in the phosphatase (0·35).

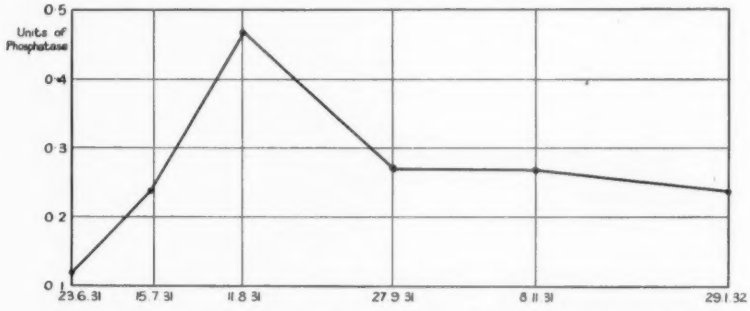
(4) By November 9 tibial hæmorrhage was absorbed and there was definite lamination of the femoral hæmorrhages.

From these and other data which cannot be debated here, it seems fairly clear that the plasma phosphatase is intimately concerned with calcification and bone formation. If time had permitted we had hoped to show a chart of some fracture cases showing the rise of the phosphatase with the throwing out of callus and the fall coincident with absorption and complete healing.

The explanation of the high phosphatase in rickets and the low phosphatase in scurvy may lie in the characteristic bone lesions in these conditions.

Whereas in rickets the distinctive character is the broad area of osteoid tissue formed at the epiphyseal junction, the increased vascularity of the cartilage and the broad band of the proliferated cartilage cells, in scurvy the most striking change is the great inhibition of osteoblastic bone growth, the diminished vascularity of the cartilaginous area, and although what does take place occurs in a normal and orderly manner the cartilage cells are greatly reduced in size and number.

Increased plasma phosphatase—cause or effect.—All the work which has been done on this subject leads one to believe that the high plasma phosphatase is produced in attempted compensation for the bone lesion—although there is a rough correlation between the severity of the disease estimated clinically and radiologically, and the level of the phosphatase. The fact that the phosphatase remains high, long after the serum calcium and the inorganic phosphorus have returned to normal, and even after the X-rays have revealed normal bone formation, suggests that the plasma phosphatase offers a more delicate test of an abnormal calcium-phosphorus metabolism than those previously mentioned.



A. L. Scurvy. Chart showing changes in plasma phosphatase. (See text.)

Whether these findings can be put to any practical purpose in diagnosis and prognosis remains to be seen, although even if they do not, it is an interesting physiological mechanism which has helped to elucidate one of the most difficult problems in pathology. It is also clear that in considering the pathology of bone diseases, a third factor must now be taken into account as well as the serum calcium and the inorganic phosphorus, and one which is liable to much wider variations from the normal than either of these two constituents of the blood.

In conclusion we should like to take this opportunity of thanking the staff of the Infants Hospital and of the Princess Louise Hospital and other friends who have so kindly put their cases at our disposal.

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[*January* 22, 1932.]

Three Cases of Obstruction of the Œsophagus.—J. N. O'REILLY, M.B. (for Dr. DONALD PATERSON).

The following three cases, illustrating the principal non-traumatic forms of œsophageal obstruction, were admitted to the Hospital for Sick Children, Great Ormond Street, within a period of two months last year.

I.—Baby D., 8 days old, admitted on account of vomiting during or immediately after each feed since birth. The food came back through the nose and mouth, and the expulsion was quite effortless.

On examination there was marked adenoid hypertrophy with much mucus in the nose and throat. The abdomen was distended and succussion splash was easily obtained.

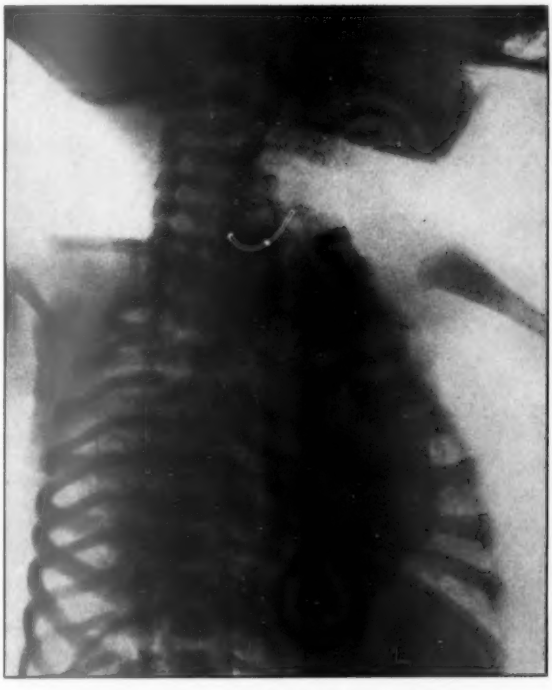

FIG. 1.—Congenital atresia of the œsophagus.

Several attempts at feeding were made but regurgitation occurred immediately and the child became cyanosed. A small stomach tube was passed but could not be pushed further than a distance of 4 in. from the lips. A skiagram after an opaque feed showed obstruction of the œsophagus at the level of the third dorsal vertebra. There was no evidence of tracheo-œsophageal fistula (fig. 1).

The baby collapsed and died twenty-four hours after admission. No post-mortem was obtained. The diagnosis made was "congenital atresia of the œsophagus."

II.—Vera T., aged 4½ years, admitted on account of vomiting. Vomiting of solid food had occurred ever since weaning. At times she seemed able to retain a biscuit

or a small piece of bread, but usually all solid food and occasionally liquids were brought up immediately after being swallowed. The child herself complained that the food seemed to stick in her chest. For the nine months previous to admission the condition had been getting worse and on admission even fluids were at times rejected.

Nothing abnormal was found on physical examination, except a certain degree of wasting. Radiologically, the œsophagus was found to be markedly narrowed at the level of the seventh dorsal vertebra. Seen on the screen, solids failed to pass at all and liquids were held up for a time and then slowly trickled through.

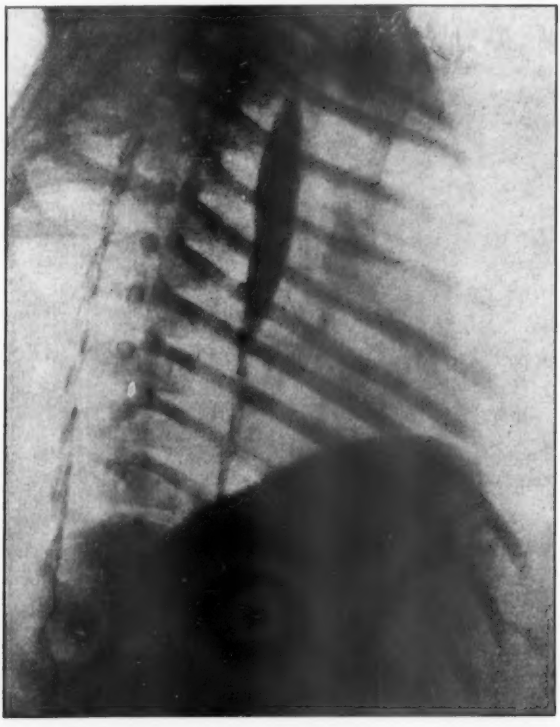

FIG. 2.—Congenital narrowing of the œsophagus at the level of the 7th dorsal vertebra.

The vomiting persisted while the child was in hospital, all solids and some fluids being brought back. A soft mercury bougie was passed in an attempt to overcome the obstruction. It was found to be difficult and at times impossible to pass this more than about 7 in. from the teeth. Once past this point, however, there was no difficulty in pushing it down about another 7 in. The vomiting was always worse after this manipulation and the attempt at dilatation was abandoned after a few days.

The child was then fed on fluids only. After a few days all vomiting ceased. Semi-solids, and finally solids, such as mince, were added to the diet, each addition being made with care. After a few weeks the child was discharged having vomited only a few times during the last two weeks of its stay. The diagnosis was "congenital narrowing of the œsophagus" (fig. 2).

III.—Norah P., aged 11½ years, was admitted to hospital with a six months' history of vomiting after every meal.

On physical examination.—Nothing of note found, except some pallor and evidence of considerable loss of weight. No family history of functional or organic nervous disorder.

Skiagrams showed dilatation of the œsophagus with closure of the cardia (fig. 3). On the screen it was found that food remained in the œsophagus for about five minutes before passing into the stomach. Mobility of the upper part of the œsophagus was good.

A mercury bougie was passed once daily before the mid-day meal. After the first day of treatment there was no further vomiting. The child was taught to pass

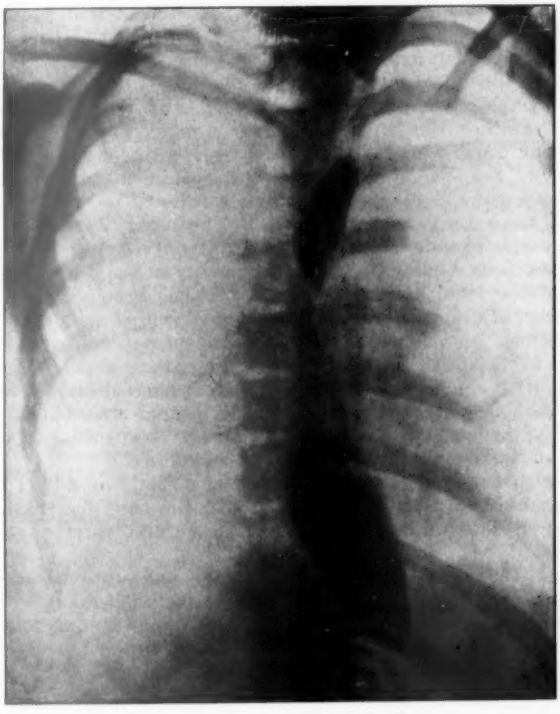

FIG. 3.—Achalasia of the cardia.

the bougie herself, and was then discharged home to continue treatment. Unfortunately the treatment was neglected at home and the vomiting commenced again. She was admitted to Cheyne Hospital in September, and was there found to be vomiting and losing weight. During her first week in hospital she lost 5 lb. She was very depressed at the prospect of using the bougie again. It was found, however, that if she was made to sniff saline up her nose into the naso-pharynx, it caused her to cough and vomit up about half an ounce of mucus and saliva. After

this she was able to take her food normally. She gained 24½ lb. in two months of this treatment. The diagnosis was "achalasia of the cardia."

I am indebted to Dr. Wilfred Sheldon for the account of this patient since leaving Great Ormond Street.

Comment.—These cases illustrate the three principal causes of vomiting of undigested food found in children.

The condition of congenital atresia of the œsophagus (Case I) has been described with a full review of the literature by Cautley [1]. Analysis of the records of the Hospital for Sick Children shows that there were six such cases among about six thousand autopsies [2]. The condition is due to a developmental defect. Keith and Spicer [3] ascribe it to irregular growth of the tracheo-œsophageal septum. Not infrequently there is a communication between trachea and œsophagus. No such fistula was found in the case described, although autopsy was not performed. On clinical and radiological grounds it may be assumed that probably no fistula existed.

The form of obstruction found in Case II has been described, with a review of the literature, by Sheldon and Ogilvie [4]. In ten cases studied by them they found the following symptoms: Wasting, constipation, and regurgitation of undigested food were noticed either from birth or from some period during the first year. The symptoms became worse when the child was tried with solid food. Radiologically, there was delay of the opaque bolus about the level of the seventh dorsal vertebra. Two of their cases came to autopsy. The first was found to have a fibrous structure at the level of the seventh dorsal vertebra. The other, having been dieted very carefully, had complete remission of symptoms for twelve months before death, which was due to tuberculous meningitis. No abnormality of the œsophagus was found.

The cause of the condition is not known. It has been ascribed to achalasia of the middle portion of the œsophagus. In the cases which have come to autopsy no evidence of this has been found. Another cause suggested is that the œsophagus develops slowly to its full size. The condition appears to be a self-limiting one, and it is consistent with the absence of pathological findings in the recovered case that recovery is due to growth of the œsophagus to normal size. It is also consistent with the clinical finding that the most successful treatment appears to be the administration of fluids and semi-solids over a long period.

Dilatation with a mercury bougie was tried in several cases by Dr. Sheldon, but was rather less successful than the fluid diet. In the case described, the child was definitely worse after dilatation—no doubt because a traumatic spastic element was being superadded.

Achalasia of the cardia is rare in infants and uncommon even in later childhood. Case III, however, appears to come into the group. The child was treated with success by a mercury dilator and relapse was due solely to abandonment of this. The alternative treatment devised by Dr. Sheldon suggests that there was a psychological element in the case. The skiagram, however, appears to prove that this was not the whole condition. Pathologically the lesion is said to be an inflammation and degeneration of Auerbach's plexus. Such a condition was found by Cameron at autopsy of a child with œsophagectasia, and has been described by others in adults.

I wish to thank Dr. Donald Paterson, under whose care these patients were, for permission to report them.

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Discussion.—Dr. F. PARKES WEBER said he believed that cases of achalasia of the cardia were not so extremely rare in children as was formerly supposed. He thought that the doctor was sometimes wrongly led to the supposition that the symptoms were partly due to a psychic cause.

Dr. POYNTON said with regard to the nervous aspect of achalasia, that some years ago a child aged between 7 and 10 years had been brought to him with symptoms of very severe spasm of the œsophagus. These had followed a rescue from a torpedoed vessel. The attacks were so severe that several times the child fell down and was thought to be dead. She died in one these attacks. The trained nurse told him that the spasm was often relieved by vomiting, and that in order to induce this, she (the nurse) would put her finger at the back of the child's throat and could actually feel it grasped by the spasm. The shock of the calamity at sea seemed to be the starting point of this extraordinary condition.

Dr. LEONARD FINDLAY: Œsophageal stenosis is a subject requiring all the elucidation possible. While œsophageal atresia is very generally known, and the error of development which causes it, well understood, congenital stenosis, on the other hand, is not generally appreciated, and its cause is even less well understood. Although there are over eighty records in the literature of congenital stenosis in childhood, in comparatively few do we know more than that there was an obstruction and where this was situated. This is owing to dearth of post-mortem evidence, and the infrequency with which endoscopic examination has been carried out. In the great majority of cases reliance has been placed on X-ray examination and the passage of a bougie. By neither of these means of investigation can any help in the elucidation of the nature of the obstruction be obtained. The X-ray appearances are identical, whether the obstruction be due to spasm, to the presence of a membranous diaphragm, to fibrous thickening of the wall, or to a narrowing of the lumen.

It has not infrequently been concluded that because the associated dysphagia has suddenly and completely disappeared after the passage of a bougie, the obstruction must have been spasmodic. The same result, however, may happen when the stricture is due to the presence of a membranous diaphragm. J. P. Clark[1] records how during an endoscopic examination he saw at the entrance to the œsophagus a membrane which ruptured immediately when the end of the instrument came in contact with it. This shows the unreliability of any opinion formed on the blind passage of a solid instrument.

From the literature I have been able to trace about twenty-three cases in which it was possible to express a definite opinion regarding the nature of the lesion. Of these, fifteen were post-mortem findings, five were endoscopic observations, and in three an operation revealed the true state of matters. The lesions discovered in these cases were (1) a simple narrowing of the lumen for a short distance of the length of the œsophagus, (2) a membranous diaphragm, (3) a fibrous or a fibro-muscular thickening of the wall of the gullet, and (4) spasm of the circular muscular fibres. Simple narrowing of the lumen and a membranous diaphragm, both of which are frequently considered analogous to atresia, were present at the most diverse levels of the viscus. This surely points to a different ætiology in stenosis and atresia. A more probable explanation is to be found in defective or perverted vacuolation of the wall of the œsophagus. During the process of development of the gullet, increase in the lumen takes place by vacuolation of the wall; the vacuoles gradually enlarge and finally rupture, causing a relative increase in the diameter of the viscus. It also seems possible that by the persistence of some portion of the wall of the vacuole, or through the absence of vacuolation for a short distance, a valve-like deformity (membrane) would result.

Severe Acute Hæmoglobinuria in a Boy.—F. PARKES WEBER, M.D.

Patient, R. V., aged 3 years, well-developed boy, admitted to hospital on November 27, 1931, with the history that on November 25, when taken out for a walk, he felt unwell, and in the evening, after his return, his urine was noticed to be red. All the urine subsequently passed before his admission to hospital had the same appearance. On admission he was still passing urine of the colour of "fruity" port wine, clear, and giving the spectrum absorption bands for oxy-hæmoglobin

[1] *Med. Record*, New York, 1911, lxxx, 600.

(positive benzidine reaction); in the very scanty sediment there were a few erythrocytes and no tube-casts. The boy looked pale. Temperature 103° F.; pulse about 130. No other signs of disease noted; no Raynaud's symptoms.

Family history.—An only child; mother died when he was born; father suffers from a mental depressive condition, and is at present a voluntary inmate at a mental hospital. The boy's blood-serum and his father's both give a negative Wassermann reaction.

The boy seems previously to have enjoyed good health. No evidence of being overtired or chilled when attack of hæmoglobinuria commenced.

Blood-count (November 27): Hb. 24%; erythros. 1,320,000; C.I. 1·0; leucos. 25,450 (eosinos. 3%; myelos. 2%; poly. neutros. 59%; lymphos. 32%; monocytes 4%); considerable anisocytosis; no poikilocytosis and no marked polychromasia; some of the erythrocytes contained Jolly bodies; four normoblasts and one megaloblast were seen whilst counting 100 leucocytes. In the evening after this blood-count the child was given a blood-transfusion of 200 c.c.

Next morning the urine (of a red-brown tinge) was of specific gravity 1021; acid; turbid with urates; still giving a positive benzidine reaction for hæmoglobin, and containing granular and hyaline casts, some leucocytes and a few erythrocytes. Blood-count: Erythros. 2,540,000; leucos. 12,300; thrombos. 457,200 to the c.mm. of blood. Temperature 102·6° F. at 7 a.m. But by about midday the temperature had come down to 99° F. and the child hardly looked ill; there was no purpura or erythema; no icteric tinge of skin or sclerotics; the urine gave a negative reaction for hæmoglobin.

No enlargement of spleen or liver was ever detected. By ophthalmoscopic examination fundi oculi appeared normal. "Fragility" of erythrocytes slightly above normal, hæmolysis commencing with the 0·52% sodium chloride solution (complete with the 0·44% solution). No worms or their ova found in fæces.

November 30.—Urine free from albumin, sugar and tube-casts, but contained decided excess of urobilinogen and urobilin. Afterwards granular and hyaline casts—and sometimes leucocyte casts—were present together with some leucocytes and a very few erythrocytes. December 17: urine free from all abnormal constituents.

Blood-count (December 3): Hb. 54%; erythros. 3,012,000; C.I. 0·9; leucos. 10,650 (eosinos. 12%; poly. neutros. 43%; lymphos. 40%; monocytes 5%); many reticulocytes present; no nucleated red cells. December 11: Hb. 71%; erythros. 4,500,000, leucos. 8,800; reticulocytes not above the normal number (about 0·5% of the erythrocytes). January 15, 1932: Erythrocyte-count again 4,500,000; leucos. 9,200 (12% of which again were eosinos.).

The hæmoglobinuria, as in most cases, was accompanied by leucocytosis, and was followed by signs of renal irritation. It lasted too long for a classical attack of paroxysmal hæmoglobinuria *a frigore*, and there is no evidence of syphilis. On December 28, when the boy had recovered, an iced cold foot-bath of ten minutes' duration failed to produce any abnormal symptoms whatever. The Donath and Landsteiner test (as given by O. Naegeli, *Blutkrankheiten*, fifth German edition, 1931, p. 671) for an auto-hæmolysin in patient's blood-serum has been carried out by Dr. W. Weisswange on January 20, 1932, with negative result.

No toxic or other cause for the hæmoglobinuria has been discovered, and I suggest that the case may represent the acutest form of Max Lederer's "acute hæmolytic anæmia" (*Amer. Journ. Med. Sci.*, 1930, clxxix, 228), and that the case of the boy, aged 11 years, whom I showed at the Clinical Section on October 9, 1931 (*Proc. Roy. Soc. Med.*, xxv, 15), was a more characteristic example without any hæmoglobinuria. In some of the cases referred to by Lederer there was hæmoglobinuria.

Case showing effect of Nirvanol on Subacute Rheumatism.—BERNARD SCHLESINGER, M.D.

History.—H. S., a boy, aged 7½ years, first came under observation in 1929, having suffered from chorea and subacute arthritis during the preceding three weeks. Mitral stenosis was present. There were also numerous large and painful nodules and intermittent pyrexia and tachycardia. Nirvanol was given for ten days and omitted when the rash appeared. Chorea disappeared, temperature and pulse-rate dropped to normal, and nodules rapidly diminished in size and number. Acute tonsillitis, followed by relapse of rheumatism with slight pericarditis, return of chorea and many fresh nodules. Pyrexia and tachycardia persisted after acute attack. A second course of nirvanol, administered for eleven days, cured the chorea and brought the pulse-rate and temperature down to normal again. By the end of two months most of the nodules had again disappeared but several small ones were still present; the pulse-rate also tended to rise again. A third course of nirvanol again brought down the pulse-rate without producing much reaction and little effect on the blood. Two months later, a fourth course of nirvanol brought down the pulse-rate for a short period. A fifth course two months later produced no reaction and was without effect. The child was discharged to a convalescent home with very few nodules still present, having gained 6 lb. during his thirteen months in hospital. He remained at a special rheumatic heart convalescent home for over six months and when seen on discharge still had one or two nodules on his knees.

Remained well during 1931 except for slight return of chorea in October.

Present state.—No signs of active rheumatism. Heart: Mitral stenosis and no advance of disease during the last two years. No tachycardia, no pyrexia, nodules absent and child gaining weight. Sedimentation rate 4.

Discussion.—Dr. GERALD SLOT said that he had examined Dr. Schlesinger's patient and had come to the conclusion that he was still suffering from active chorea, in spite of the long therapy. The results in this case were similar to those which he had himself experienced with the use of nirvanol, and he must confess that he was not impressed with its efficacy. Sir Archibald Garrod had said that one could cure chorea in six weeks with any drug, provided one gave it in doses that were toxic, and he believed that the use of nirvanol merely expedited a toxic process.

With regard to the nodules, he looked upon them as usually indicating a serious prognosis. He had tried on many occasions to remove these nodules for microscopic purposes, and had failed. When cut down upon, that which felt hard and firm appeared to be a collection of fluid and fibrin, and disappeared when attempts were made to remove it. There was of course another type of nodule which was harder and easier to remove if previously transfixed, but most of those with which he had dealt were impossible to remove *in toto*.

Dr. W. R. F. COLLIS said he believed nirvanol to be a thoroughly dangerous drug. He had seen a patient die from its effects in a case of acute rheumatism, and in another case the patient had become mentally deranged by its administration. He did not think that its employment as a therapeutic drug in cases of chorea was justifiable. Dr. Schlesinger's suggestion, however, that it was anti-rheumatic, was interesting, and the case which he had shown certainly bore out his contention, but one case was not sufficient basis for judgment and nothing more could be said until a large series of cases had been produced.

His experience with regard to the removal of rheumatic nodules did not agree with that of Dr. Slot. He himself and others had on several occasions successfully excised these nodules.

Dr. E. STOLKIND said that long ago he had treated cases of chorea with salicylates, bromides, etc., but later had tried to eliminate drugs and give rest, good food and psychotherapy (persuasion). The results were the same as with the drugs. Lately, in addition to the rest, he had, when necessary, given arsenic and iron.

Dr. LEONARD FINDLAY: In my experience nirvanol has never seemed to have the slightest effect in chorea; in fact, of a series of cases of chorea treated by different measures, those in

which nirvanol was employed had the longest average duration. It is a striking fact that even although salicylate of sodium behaves like a specific in rheumatic arthritis, it is not possible to demonstrate any beneficial effect from its administration in any other rheumatic manifestation. Hence one would require more definite evidence than mere slowing of the pulse to prove any anti-rheumatic power of nirvanol.

I do not agree with Dr. Slot that rheumatic nodules cannot be excised for histological examination, and his failures, I think, are due to a misunderstanding of their nature. True, there is no more remarkable difference than the apparent stony hardness of the nodule when palpated *in situ* over a bony prominence and its almost complete impalpability when excised for the purpose of a biopsy, but if the excised tissue is examined there will not remain any doubt regarding the possibility of performing the operation. This difference in the two states I ascribe to the fact that in the formation of the nodule, just as in all rheumatic lesions, œdema plays a prominent part, and that during the process of excision the capsule has been injured and has permitted of the escape of much fluid.

Dr. SCHLESINGER (in reply) said that the stimulating discussion had given him further encouragement in pursuing this special form of treatment. Most of the speakers had tended to wander from the subject and concentrate on chorea, whilst the inability or otherwise of various observers to remove nodules during life, although interesting, was beside the point. The general opinion of the meeting appeared to be that nirvanol was a dangerous drug. This was only so if given in the wrong type of case, and then the danger lay not in any toxic property of the drug, which never appeared in the doses recommended, but in its liability to cause a temporary exacerbation of the rheumatic process before the nirvanol reaction took place and the desired improvement could be expected. This peculiar property might thus cause the flare-up of a smouldering pericarditis or enhance the mental disorganization in certain severe cases of chorea. Nirvanol should therefore not be used in acute rheumatic fever, acute carditis, or acute chorea in which the mental symptoms were the predominant feature. If these rules were observed, the drug was perfectly safe and in certain cases of subacute smouldering rheumatism with active carditis it had, in his (the speaker's) hands, proved remarkably successful when other measures had failed, and the patient had slowly been losing ground. With this in view he had shown this case to-day, as it seemed to demonstrate an apparent anti-rheumatic effect of the drug on a particularly severe and obstinate case. The beneficial anti-rheumatic effect was not only seen here in connection with the pulse-rate, as Dr. Findlay had remarked, but also on the temperature, nodule formation, gain in weight, and general well-being of the child.[1]

Fibrocystic Disease of the Ulna.—CECIL P. G. WAKELEY, F.R.C.S.

H. V., aged 8 years, fell down on his left elbow in November, 1931. He complained of pain in the elbow and as it was noticed that the region of the joint was swollen, his parents took him to a doctor. *On examination.*—Diffuse, hard swelling of upper end of ulna, not attached to the skin. Movements of the elbow-joint full and free, and in no way limited by the swelling. Skiagrams showed distinct cystic expansion of upper end of the ulna to the extent of 2 in.

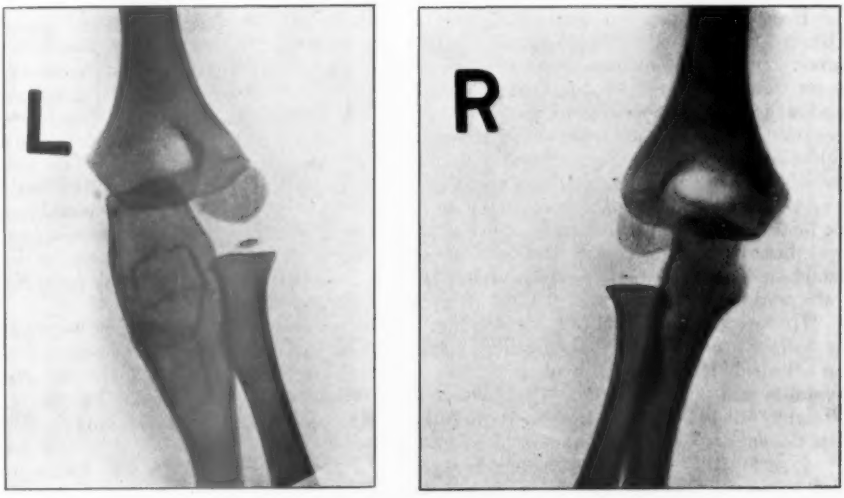
Dr. M. F. Hope, of Andover, removed a piece of the cyst wall for section.

Pathological report (Royal Victoria Hospital, Netley):—

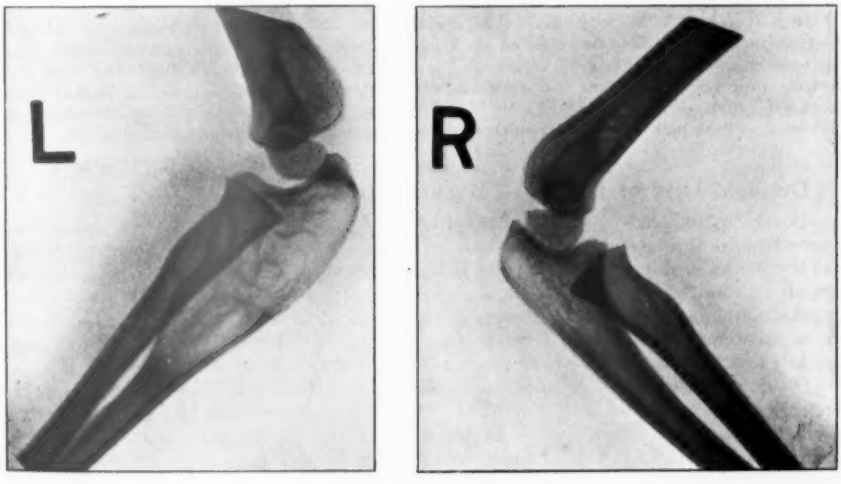
In view of the small amount of material supplied, a complete diagnosis is not possible. It can be definitely stated, however, that the condition is not myelomatous. The greater part of the tissue is solid hyaline material, probably the result of degeneration in a neoplasm: and occasional groups of cells are found which appear to be of neoplastic origin. The microscopic evidence tends to support that of sarcoma—probably a slow-growing fibro-sarcoma.

In spite of this report, I am inclined to think the condition is not malignant, but is due to fibrocystic disease of the bone.

[1] The early history of this case was originally published in "Recent Advances in the Study of Rheumatism" (J. & A. Churchill, London, 1931).



Antero-posterior view.



Lateral view.

FIBROCYSTIC DISEASE OF LEFT ULNA.

The fibrocystic disease in the left ulna is well shown. The right forearm bones are shown for comparison. (Skiagrams by Dr. Graham Hodgson.)

Heart Disease with Abdominal Systolic Murmur. Coarctation of Aorta.—ALAN MONCRIEFF, M.D.

Harold E., aged 6 years. Admitted to the Hospital for Sick Children, Great Ormond Street, under Dr. Hugh Thursfield in October, 1929, with acute rheumatic fever. Only previous illness was whooping-cough, and the symptoms of the rheumatic fever were of a few days' duration. Cardiac involvement developed while in hospital, and a green streptococcus of the mitis type was grown from his blood. The heart became very much enlarged, with a canter rhythm, and at the apex systolic and mid-diastolic murmurs were heard. Shortly after the heart became involved he was found to have a loud systolic murmur all over the abdomen, best heard over the liver, and there was also a systolic thrill to be felt over the liver. The murmur could not be heard over the femoral arteries. Early in January, 1930 a loud systolic murmur was heard over the aortic area, conducted up into the neck, and a systolic thrill could be felt in the vessels of the neck. The boy made an excellent recovery from his very acute illness and went to West Wickham for eight months.

His present condition is much the same as before as regards the very widespread systolic murmur which can be heard down as far as the umbilicus. Skiagrams show an enlarged heart, especially affecting the left ventricle. Blood-pressure is 150 mm.Hg systolic and 100 diastolic. No evidence of collateral circulation can be found. Weight 46 lb. (normal for age, 45¼ lb.). He is at present attending under Dr. Cockayne, to whom I am indebted for permission to show him.

POSTSCRIPT.—At the meeting, evidence of collateral circulation was found in the region of the left scapula. This seems to make the diagnosis of coarctation of the aorta the most likely one. Correct blood-pressure—150 (systolic), 100 (diastolic).

Dr. F. PARKES WEBER said that, as evidence of compensatory collateral arterial circulation had now been detected on the back, there could be no difference of opinion that the case was one of stenosis (coarctation) of the aortic isthmus. He suggested that the systolic murmur heard over the upper part of the abdomen might possibly be due to a so-called "mycotic aneurysm" caused by infection of the arterial walls with *Streptococcus viridans*—which had indeed been cultivated some time ago from the circulating blood.

Dermoid Cyst of Brain.—J. WILKIE SCOTT, M.D.

D. W., male, aged 2½ years. Brought to hospital October 31, 1931 on account of backwardness and loss of power in right arm. Head has always been large from birth, but there was nothing very abnormal in development. Talked at 1 year; walked at age of 18 months, but clumsily, and frequently fell down. In April, 1931, he was knocked down and sustained bruising of right side of head and face; was very dazed for a fortnight. From that time he has not walked or talked, and there has been marked loss of power in the right arm; there has also been great irritability.

On examination.—He is undersized but well nourished, can stand with support, understand simple directions, and is bright and friendly; makes a feeble attempt to say single words. Head large, circumference 22 in., hydrocephalic in shape with some rachitic bossing; rickety teeth; exophthalmos more marked on left side; some limitation of movements of left eye in all directions and ptosis; discs very pale, though vision for objects and recognition is good. Still marked weakness of right arm. Cerebrospinal fluid normal, except for slight increase of globulin; total protein 0·091. X-ray examination (Dr. R. A. C. Rigby) shows presence of numerous teeth in the neighbourhood of the sella turcica and more posteriorly; also a shadow suggesting outline of a cyst (figs. 1 and 2).

There has been gain in weight, and decided improvement in mental condition since admission to hospital.

FIG. 2.—Dermoid cyst of brain. Lateral skiagram.

FIG. 1.—Dermoid cyst of brain. Antero-posterior skiagram.

Suprapituitary Cyst.—J. H. GIBBENS, M.R.C.P.

Girl, aged 5, attended hospital first in December, 1930. For the previous year she had had attacks every two to four weeks of headache and vomiting; these attacks always came at night, usually at about 2 a.m., and lasted all night. First she complained of left-sided headache, which became worse and worse through the night, to be relieved finally by profuse vomiting. After this she went to sleep and woke up well: in the intervals her health was excellent. No ocular symptoms or fits at any time. No thirst or polyuria.

On examination.—Left pupil dilated and insensitive to light directly, although reacting briskly consensually. Right pupil of moderate size, reacting to light briskly both directly and consensually. Left eye: no perception of light. Left optic disc very pale, pallor not obviously consecutive to œdema: retinal arteries considerably reduced. Right eye: Vision difficult to determine, but apparently considerable loss of the temporal field. Right fundus: Chronic papillœdema with slight reduction in size of the retinal arteries. No definite left external rectus palsy, but on looking to the left, the left eye does not move in quite the same axis as the right: it tends to move outwards, slightly upwards, with slight rotation. All other eye movements normal. No nystagmus, no exophthalmos. All cranial nerves normal. All reflexes in arms and legs normal. No signs of cerebellar disease. No obesity or genital dystrophy. Urine: No polyuria: no albumin or sugar.

X-ray examination (17.12.30) showed partial destruction of sella turcica, especially of posterior clinoid processes. No suprasellar or intrasellar calcification.

Diagnosis: Suprapituitary cyst.—The child was admitted for operation, and was seen by Sir Percy Sargent, who, however, did not advise operation but suggested deep X-ray therapy. This was begun in February, 1931.

29.4.31.—Three attacks of vomiting in nine weeks. Left pupil now reacts directly to light, but slowly and to only a slight extent: there is perception of light in left eye. Condition of left fundus unchanged, but right fundus now shows secondary atrophy.

27.5.31.—No headaches or vomiting.

22.7.31.—One attack of headache, vomiting and drowsiness. No change in the physical signs. X-ray examination: Evidence of increased intracranial pressure—separation of the sutures and digitation of the skull. Posterior clinoid processes show increased destruction.

11.11.31.—During the last month, three attacks of very severe headache and vomiting, lasting 12 to 24 hours.

A total exposure to deep X-rays of fifteen hours has been given over the last nine months: at first the exposures were given twice weekly for half an hour, then once weekly, with occasionally a month's complete rest. Never any symptoms of overdosage.

10.1.32.—Headaches now becoming much more frequent. X-ray examination shows increased separation of the sutures and advanced destruction of the sella turcica. The discs show secondary optic atrophy more advanced on the left side.

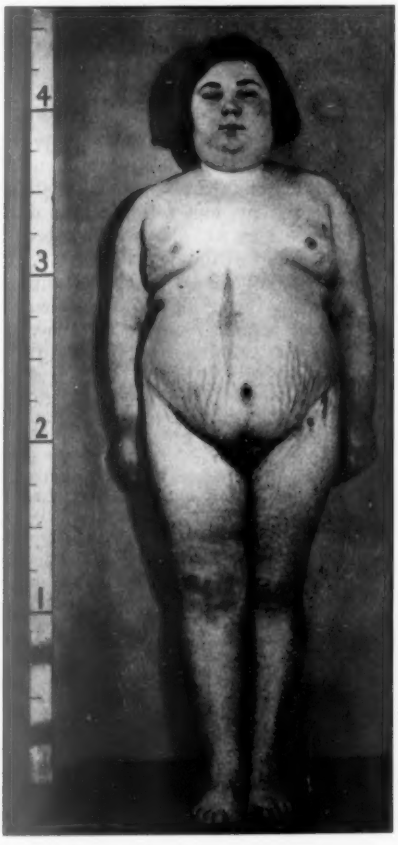
Obesity and Hirsutes of ? Adrenal Origin.—R. W. B. ELLIS, M.D. (for Dr. A. G. MAITLAND-JONES).

D. B., a girl, aged 12 years.

Both parents short and stout. Nine other children alive and well; one died after measles; one still-born. Patient has been plump since infancy, but not excessively so. Weight at 8½ years of age was 61 lb. Was seen as out-patient at 10 years of age on account of obesity and was dieted; pubic and axillary hair began to appear at this time. At 11 years of age the obesity increased very rapidly especially in the region of the face, neck, shoulders and abdomen; striæ appeared in

the loins, while the pubic hair began to assume the male distribution. Skiagram of pituitary fossa at this time showed no abnormality. No restriction of visual fields; discs normal; no evidence of increased intracranial pressure. Patient has never menstruated though pubic hair has now been present for two years.

July, 1931.—Admitted to hospital on account of pain in left loin, unaccompanied by frequency or dysuria. Urine was then normal. Physical examination showed



D. B., showing male distribution of pubic hair and striæ atrophicæ on thighs, abdomen and breasts.

very marked obesity; weight 104 lb., height 4 ft. 4 in., very rubicund facies and presence of pubic hair extending up to umbilicus with much fine dark hair on back of shoulders and arms. Blood-pressure 135/95 to 150/80. Temperature and respiration normal. Pulse 90 to 120 at rest. Aortic second sound accentuated. Pyelograms appeared normal. Blood-urea 21 mgm. %. Urea concentration and phenol sulphonephthalein tests normal. Blood phosphatase 0·435. Inorganic phosphorus 2·75 mgm. %.

29.7.31.—Cerebrospinal fluid normal; pressure 210 mm. Wassermann reaction negative. Discs, visual fields and skiagram of sella turcica normal. Electrocardiogram showed normal rhythm; no relative preponderance; T. 1 and T. 2 upright and very tall; P. 3 and T. 3 inverted; the latter "probably due to change in the heart axis following on obesity and a high diaphragm." Orthodiagram showed a high aortic knuckle but no cardiac enlargement. Fluid intake and output normal.

In view of the clinical appearance indicating adrenal hyperplasia, a laparotomy was performed by Mr. Neligan. No tumour was found, but owing to the extreme obesity a satisfactory exposure could not be obtained.

After discharge from hospital, severe left-sided renal colic with hæmaturia occurred; skiagram showed a large calculus at the lower end of the left ureter, which was passed spontaneously.

During the past six months the obesity has rapidly increased. Weight now 134 lb. Height stationary. Striæ atrophicæ present not only in the loins but vertically down the abdomen, around breasts, and on shoulders and thighs. The hirsutes has increased and the patient has a few coarse dark hairs on the chin and upper lip, and around the nipples and umbilicus. The breasts are moderately large and pendulous but show no evidence of functional activity. Intellectual development is normal for her age; no evidence of sexual precocity; emotionally immature. She has had a persistent tachycardia (over 100) and somewhat dilated pupils for the past six months, and there has been a recurrent vulvo-vaginitis with some slight discharge. No polydipsia, polyuria, or abnormal bleeding. Blood-pressure (12.1.32) 140/90 to 150/95. Pulse, 112.

Blood-count: (24.9.31).—R.B.C. 4,500,000; Hb. 80%; C.I. 0·88: W.B.C. 11,600. *Differential*: Polys. 75%; eosinos 0·5%; small lymphos. 11%; large lymphos. 4%; large hyals. 9·5%.

Sugar Tolerance Test. (25.8.31):—

Fasting	·097%	1½ hours	·080%
50 g. glucose ...	p.o.	2 hours	·086
½ hour	·086	2½ hours	·116
1 hour	·116	3 hours	·100

[*The report of other cases shown at this Meeting will be published in the next issue of the* PROCEEDINGS *of the Section.*]

Section of Pathology.

[*January* 19, 1932.]

The Primary Lung Focus of Tuberculosis in Children.[1]

By JOHN W. S. BLACKLOCK, M.D.

(From the Departments of Pathology of the University and the Royal Hospital for Sick Children, Glasgow.)

ABSTRACT.—Of 1,800 consecutive autopsies on children whose ages ranged from a few hours to between 12 and 13 years, and who lived in Glasgow and the West of Scotland, 283 (15·7%) were found to have tuberculous lesions.—Of the total tuberculous cases, the lungs or tracheo-bronchial lymph nodes were the seat of tuberculous lesions in 173 (61·1%), and of these 168 (97·1%) died as a result of the tuberculous disease.—A primary lung lesion was found in 148 of the cases classified as having the primary site of infection in the thorax. The primary lung lesion consisted of a localized patch of caseous broncho-pneumonia and in the great majority of cases was single. Calcification of these lesions was unusual and only about one-third showed evidence of surrounding fibrosis, the incidence of which increased with the age of the children.—Most of the primary lung lesions were subpleural in position and the right lung was more often the seat of such lesions than the left; the right upper lobe being most frequently involved and then, in order, the right lower, left upper, left lower, and right middle.—The tuberculous adenitis in the tracheo-bronchial glands was related both anatomically and pathologically to the primary lesion in the lungs, the involvement of the glands being secondary to the lung lesion.—In 25 of the cases considered as primary thoracic infections, no primary lung lesions were found.—In a series of cases in which the type of infection was investigated, 173 human and 3 (2·7%) bovine strains were found in cases with the primary site of the infection in the thorax.—Only human strains were obtained from cases with primary lung lesions.—These findings were in marked contrast to those found in children in the same series with the primary site of infection in the abdomen where 81·8% of the infecting bacilli were of the bovine type, and also in cases of bone and joint tuberculosis—a blood-borne infection, in which 34·6% of the causal organisms were bovine strains.—From the pathological and bacteriological evidence, it is concluded that the focus described as the primary lung lesion is indeed such, and that it is due to direct infection of the lungs through the air passages.

DURING the last seven years we have found 283 cases (15·7%) with tuberculous lesions in a consecutive series of 1,800 autopsies performed at the Royal Hospital for Sick Children, Glasgow. The ages of the children coming to *post mortem* in this series ranged from a few hours to between 12 and 13 years, and they were mostly children of poor or working-class people. The 283 tuberculous cases were classified according to what was considered the primary site of the infection as follows:—

Thoracic ...	173 (61·1%)	Cervical glands ...	6 (2·1%)
Abdominal ...	101 (35·7%)	Not found	7 (2·5%)

[1] The expenses of this work were partly paid by a grant from the Medical Research Council to whom I wish to acknowledge my indebtedness.

Four cases had double primary sites of infection, one in the thorax and the other in the abdomen. It is the primary thoracic lesions which we wish to discuss more particularly in this communication, as the results of the whole investigation are being published shortly by the Medical Research Council as a special report. The greatest number of children with primary intrathoracic tuberculous disease was found in the first year of life, though in the first three months they were rare. After the first year, the greatest number was found in the second year, and then in the third (Table I). After the third year, the number of cases in any one year was always less than half of that in the third, and for convenience the cases have been arranged in groups of three years in the Table. Nearly all the lesions had been responsible for death, only in five (2·9 %) of the 173 cases could they be regarded as accidental post-mortem findings. In only two of these five, however, which were not responsible for death, was there naked-eye and microscopic evidence that the disease was in the process of being arrested. In one of these children, aged 4 years and 1 month, the lesion in the lung was partly calcified and surrounded by fibrous tissue (fig. 6), and the glands at the root showed slight calcification with marked chronic periadenitis; in the other child, aged 5 years, a few chronic partly calcified caseous glands were found at the root of the left lung in which no primary lesion was discovered. Thus nearly all these children with tuberculous lesions in the lungs and tracheo-bronchial lymph nodes died as a result of the tuberculous disease.

TABLE I.

Age incidence, etc., of 173 cases with primary intrathoracic tuberculosis in 1,800 consecutive autopsies.

Type of case	Result of lesion	0 to 1 year	1+ to 2 yrs.	2+ to 3 yrs.	3+ to 6 yrs.	6+ to 9 yrs.	9+ to 13 yrs.	Totals	
148 cases with primary lung lesions	Caused death ...	52	37	18	15	17	6	145	148
	Not cause of death	...	...	...	2	1	...	3	
25 cases with no primary lung lesions	Caused death ...	5	3	4	7	3	1	23	25
	Not cause of death	...	...	1	1	...	...	2	
Total primary thoracic cases = 173 ...		57	40	23	25	21	7	173	
Total autopsies		1,204	235	87	135	97	42	1,800	

We have divided the 173 cases with primary intrathoracic infections into two groups:—

(1) Those in which primary lung lesions were found; (2) those in which *no* primary lung lesions were discovered.

(1) CASES IN WHICH PRIMARY LUNG FOCI WERE FOUND.

In this group there were 148 cases. The primary lung lesion was originally described by Parrot (1876) and later by Kuss (1898), E. Albrecht (1907), H. Albrecht (1909), Ghon and Roman (1913), Ghon (1916), Opie (1917), Canti (1919), Schürmann (1926), and Ranke (1928). Zarfl (1913), Ghon and Pototschnig (1918) have described very early stages of the lesion, while Gardner (1922) and Pagel (1926) have experimentally produced in animals lesions similar to those in the child.

Characters of the primary lung lesion.—In our cases the primary lesion was found to be a small localized caseous area in the lung substance (fig. 1). Microscopically it consisted of a patch of caseating tuberculous broncho-pneumonia which was fairly sharply marked off from the surrounding lung tissue, and which nearly always showed on microscopic examination some evidence of an attempt at arrest (figs. 2 to 6). Histologically a small bronchiole plugged with caseous material could often

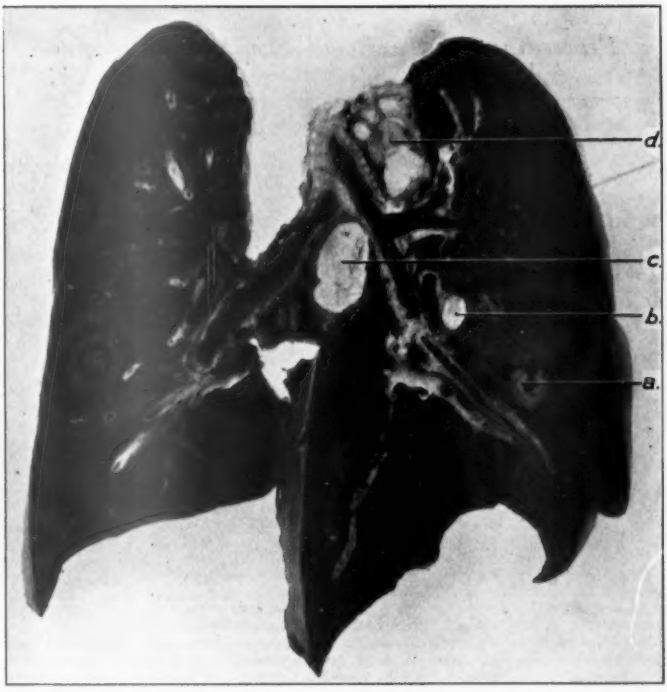


FIG. 1.—Lungs of child aged 1 year and 7 months, with primary lesion in lower part of right middle lobe. The specimen is viewed from the posterior aspect. The primary lesion (*a*) is caseous with central cavity formation. In the outer part of the lobe there is some early broncho-pneumonic tuberculosis, while scattered throughout the rest of the lungs numerous subacute miliary tubercles are present. A small caseous broncho-pulmonary gland (*b*) is present at the root of the middle lobe and the right inferior (*c*) and the right superior (*d*) glands related to the primary lesion are enlarged and caseous. No diseased glands are present at the root of the left lung.

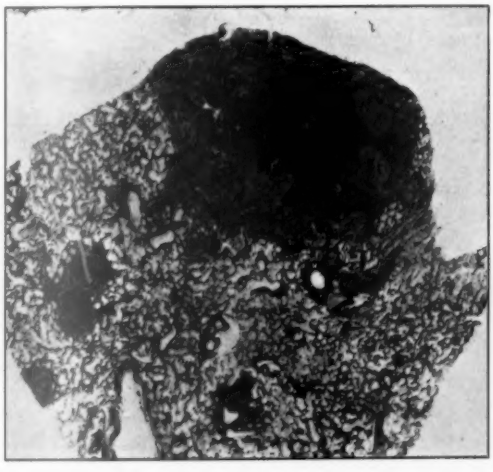


FIG. 2.—Child aged 7 months; a primary lung lesion at an early stage. The lesion is situated immediately under the pleural surface and is fairly sharply demarcated from the surrounding lung tissue. Numerous small vessels and bronchioles are involved in the lesion, the central part of which is caseous and shows early softening, while in the outer part there is a zone of cellular reaction. The small foci in the lower part of the figure are secondary subacute miliary lesions. Hæmatoxylin and eosin × 6½.

FIG. 3.

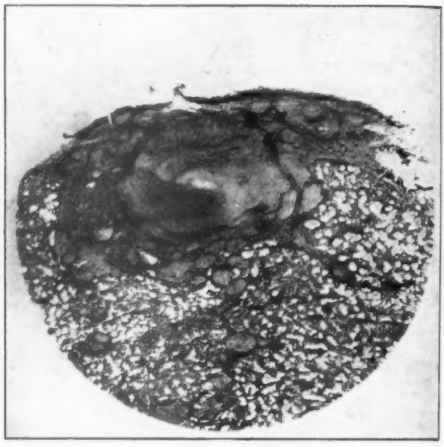


FIG. 4.

FIG. 3.—Child aged 6 months. Primary lung lesion at a slightly later stage than that in Fig. 2. The subpleural lesion is completely caseous, with, at the margin, scanty cellular infiltration and early fibrosis. The pleura over the lesion is slightly thickened and many small tubercles are found in the line of a small bronchiole, part of which is seen in the upper part of the figure. Such involvement of the peribronchial lymphatics was not often observed. Hæmatoxylin and eosin × 10.

FIG. 4.—Child aged 7 years. Caseous subpleural primary lesion at a later stage than that in fig. 3. There is well-marked surrounding fibrosis both at the edge of the main lesion and also around the numerous secondary tubercles. The caseous material in the centre is beginning to break down. Gallego's modification of Mallory's stain × 5.

FIG. 5.—Child aged 7 months. Caseous subpleural primary lesion at a later stage than that in fig. 4. The lesion is completely surrounded by fibrous tissue. Numerous subacute miliary tubercles and areas of early broncho-pneumonic tubercle are present in the lower part of the figure.
Gallego's modification of Mallory's stain × 10.

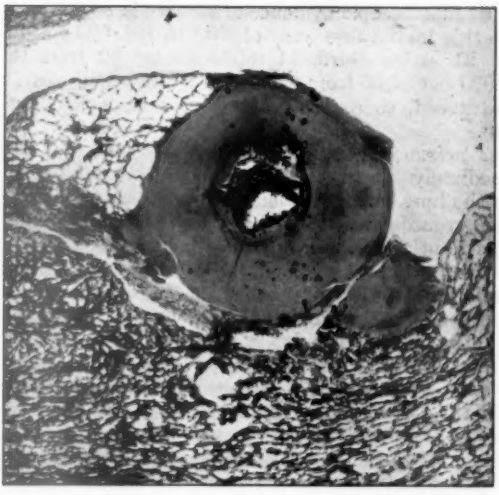


FIG. 6.—Child aged 4 years and 1 month. Died from chronic meningococcal meningitis. Section from the primary lesion which showed the most advanced evidence of arrest of any found in the whole investigation. The lesion (subpleural in position) is well encapsulated and there is only one secondary tubercle at the right edge. The central darkly-staining area is calcified and part of this has fallen out in the preparation of the section. The pleura over the lesion is slightly thickened and there is a little emphysema in the surrounding lung tissue.
Von Kossa's silver method and carmalum × 6¼.

be found leading into the primary lung lesion which, when stained for elastic tissue, showed the normal pattern and architectural arrangement of the lung tissue persisting in the consolidated area. Though a large number of primary lesions were cut in serial sections, we could never demonstrate any of them to have a vascular origin. In the majority of cases only a single lesion was present, but in a few cases where they were multiple one lesion was often found, on naked-eye and histological examination, to be older than the others. In this series of 148 cases the actual distribution of the primary lesions was found to be as follows:—

133 cases had	1 primary lesion each.
10 cases had	2 primary lesions each.
3 cases had	3 primary lesions each.
2 cases had	4 primary lesions each.
148 cases had	170 primary lesions.

The fact that the greatest proportion (89·9%) of the cases had only one primary lung lesion is difficult to explain, as a child in contact with a patient suffering from "open" pulmonary tuberculosis would inhale large numbers of bacilli and multiple lesions would be expected. Accordingly, unknown factors must be at work in determining the localization of the lesion in the lung substance. Most of the above 170 primary lung lesions were small, 140 (82·4%) being the size of a hazel-nut or smaller. In all of them caseous change had taken place, and in 52, cavities had formed. Only 11 (6·5%) showed definite macroscopic evidence of calcium deposit, while in 55 (32·4%), included in which are those showing calcification, surrounding fibrosis was noted at the site of the lesion. No completely fibrosed or calcified primary lesion was found in the whole investigation, and in this finding we are at variance with many of the Continental and American workers already quoted. The

age incidence of this macroscopic evidence of healing is of interest. One case (1·7%) out of 58 showed this in the first year of life, 15 (34·9%) out of 43 in the second, 8 (38·1%) out of 21 in the third, 11 (50%) out of 22 from the fourth to sixth year, and 20 (76·9%) out of 26 from the seventh to twelfth year. Thus, as the age of the children advanced, so a greater proportion of cases showed an attempt at arrest of the lesion.

Situation of the primary lesions.—The majority (70%) of the primary lesions were found immediately under the pleural surface while the remainder were situated deeper in the lung substance, but generally nearer one surface than another. Of the 170 lesions regarded as primary 112 (65·9%) were found in the right lung. When the lobar distribution of the lesions was investigated, the greatest number was found in the right upper lobe, and then, in order, the right lower, left upper, left lower, and right middle. In the upper lobes these lesions were most often found in the upper two-thirds of the anterior surface; the apex, however, was very seldom involved. In the lower lobes, the lower third of the posterior aspect was the commonest site, and in the middle lobe the middle third of the anterior surface.

Relation of primary foci to the tracheo-bronchial lymphatic glands.—The lymphatic glands into which the lymph from the lungs and pleuræ drain are the tracheo-bronchial. The right and left superior groups of glands situated above the main bronchi on either side drain the corresponding upper lobes. The right inferior group situated below the right main bronchus receives lymph from the right lower and middle lobes, and the left inferior group from the left lower lobe. Although this is mainly so, yet there is a certain amount of overlapping of the lymphatic territories draining into these groups due, as Most (1908) pointed out, to the lymphatics from the contiguous pleural surfaces of neighbouring lobes draining into a common deep lymphatic channel in the depths of each interlobar fissure.

For the study of the anatomical relationship of the primary lesions to the glands, we considered chiefly those cases with only one lesion. Where there were multiple lesions, either of a primary or secondary nature, scattered throughout different lobes, the anatomical relationships to the lymph nodes were not so clearly demonstrable. This was also true, to a certain extent, of multiple lesions in one lobe, as the lymph glands in these cases often showed extensive lesions on account of the greater size of the diseased area. We found in cases with only one primary lung lesion that the situation and extent of the tuberculous adenitis in the tracheo-bronchial glands was a very good indication as to the site of the primary lesion in the lung substance. Space does not permit of a complete analysis of the situation and extent of the glandular lesions in those cases with single primary foci, but in general we found that the glands on the right side showed the most advanced tuberculous disease, when the primary lung lesion was on that side, the superior group, when the primary lesion was in the upper lobe, and the inferior group, when the lesion was in the middle or lower lobes. The same was true of the glands on the left side related to primary lesions in the left lung. The tuberculous disease did not, however, long remain localized to the glands in direct anatomical relation to the various lobes, and in the great majority of cases there was spread, upwards or downwards, to the lymph nodes on the same side, and then across to the glands on the other side. In these cases with extensive glandular involvement, however, the glands in direct anatomical relation to the primary lesion in the lung always showed the most advanced pathological change. As most of the lesions were in the right lung, the glands on the right side were more often diseased than those on the left. In the few exceptional cases where the glands were not involved in accordance with their anatomical relations to the lobes of the lungs, the lesions were situated in the upper parts or interlobar surfaces of the middle or lower lobes, and probably the infection had passed through the pleural lymphatics to the lymphatic channel in the deep part of the interlobar fissure already mentioned. In our series of cases all the primary

lung lesions studied were associated with tuberculous adenitis in the tracheo-bronchial glands. It was also noted that the extent and degree of involvement of these glands were less in older children than in infants.

The pathological changes in the glands were found, on both naked-eye and histological examination, to be at the same or an earlier stage than those present in the associated primary lung lesion. By using the delicate silver nitrate test of von Kossa for the demonstration of calcium salts, we were able to show in a large number of cases that early deposit of calcium salts was present in the primary lung lesion, but not in the related glands. These observations on the age of the lung lesions and glands were of importance as they showed that the disease in the latter was secondary to the lesion in the lungs.

The important facts obtained from this study of the tuberculous adenitis associated with the primary lung lesions were that the glandular disease was most marked on the side of the lesion and very often in relation to the actual lobe in which the lesion was present. Further, the broncho-pulmonary glands situated between the branches of the bronchi to the various lobes, and also, occasionally, those within the lung substance, were always diseased on the side of the primary lesion in the lung, while those in relation to the lung on the other side showed no morbid change. In addition to these observations, we noted that the further the diseased glands were traced away from the lesion in the lung, the less intense did the tuberculous adenitis become. Thus a retrogressive spread from diseased glands, a process which we never observed, having been the cause of the primary lung lesion seemed most unlikely.

(2) Cases in which No Primary Lung Foci were discovered.

In twenty-five of the 173 cases considered as primary intrathoracic infections, no primary lung lesions were found. In all of these cases except two in which double portals of infection were present, the tuberculous adenitis in the thoracic glands was advanced and tuberculous lesions elsewhere were of a secondary nature. In twelve of the subjects, a primary lesion in the lung might have been present, as the distribution of the tuberculous adenitis in the tracheo-bronchial lymph nodes in these children corresponded to cases in which primary lung lesions were found. On the other hand, in these cases it is possible that the infecting bacilli passed through the bronchial mucosa without leaving any trace of their passage, as in the case of the intestinal or pharyngeal mucosa in subjects with tuberculous adenitis in the mesenteric or cervical glands. Most of the other workers on this subject, quoted above, state that this does not occur, and that in such cases the primary lung lesion has been missed. Until we have had further experience, however, of cases without primary lung lesions, we feel that such a view is rather extreme. In other nine children, the tuberculous lesions in the lungs were so extensive that the finding of a primary lesion was impossible. In the remaining four cases, the most advanced tuberculous disease was in the thoracic paratracheal glands, and the site of the primary infection in these cases was uncertain, though it may have been in the trachea or one of the main bronchi.

Bacteriological Findings.

From the pathological part of the investigation we have shown that the primary lung lesion was most probably the first site of the tuberculous infection, and was due to infection through the air passages. One thing remained to make the proof of this more or less complete, namely, what was the type of infecting bacillus in those lesions or associated glands? Conditions in Scotland favoured this part of the investigation, for, as Fraser (1912), Mitchell (1914), Griffith (1915 and 1930), Tulloch and his co-workers (1924), Munro and Cumming (1926) and Munro (1930) have shown, a very large proportion of tuberculous lesions

in that country were due to infection with bovine strains. As infection with that type of tubercle bacillus nearly always takes place through the alimentary system, we tried to isolate the infecting organisms from the first 216 cases with tuberculous lesions occurring in this series of autopsies, and were successful in obtaining strains from 183 cases. These organisms were typed according to their cultural characters, and also by the results they produced on inoculation into rabbits. From lesions considered as primary abdominal, 54 bovine (81·8%) and 12 (18·2%) human strains were obtained (Table II). Now, according to von Behring (1903) and Calmette (1922), practically all tuberculous infection enters the body by way of the alimentary tract. If their views are correct, and as the children

TABLE II.

Types of tubercle bacilli found in (1) *primary thoracic tuberculosis*, (2) *primary abdominal tuberculosis*, (3) *tuberculosis of bones and joints.*

Source of strain		Human		Bovine		Bovine percentage
Cases with primary lung lesions	Primary lesion	32	94	—		—
	Related glands	62.				
Cases with no primary lung lesions	Tracheo-bronchial glands	11	13	2	3	18·8
	Paratracheal glands ...	2		1		
All primary thoracic lesions		107		3		2·7
Primary abdominal lesions		12		54		81·8
Tuberculosis of bones and joints		17		9		34·6

with primary abdominal infections from which we isolated the above high percentage of bovine strains were from the same series of autopsies as those with the primary thoracic lesions, then we ought to have found a high percentage of cases in this latter group infected with bovine strains. This was not the case, however, for from subjects in whom the primary site of the infection was in the thorax, 107 (97·3%) human and three (2·7%) bovine strains were isolated from the primary lung lesions or tuberculous tracheo-bronchial glands. Only human strains were isolated from cases with primary lung lesions, while the three bovine strains were obtained from subjects in whom no such lesions were discovered (in two of these there was tuberculosis of the tracheo-bronchial lymph nodes, the right superior group being involved in the one, and the right inferior in the other, while in the remaining case one of the left thoracic paratracheal glands was the seat of the most advanced disease). Thus there was a very marked difference in the type of infection in the cases classified as primary abdominal and in those with the primary seat of the disease in the thorax. So marked was the difference that the conclusion was justified that the subjects with the primary seat of the tuberculous disease in the thorax were not infected by way of the alimentary system. Further, in view of the fact that 97·3% of the thoracic cases were infected with human strains, we concluded that the path of infection was directly through the air passages. The three thoracic cases in which no primary lung lesions were found, but which were infected with bovine strains, may appear to be against this conclusion. This, however, is not necessarily so, for, as Griffith (1930) has pointed out, "open" respiratory carriers of the bovine type of bacillus are relatively common in Scotland where they constitute 3·8% of patients expectorating viable tubercle bacilli, as against 1% in England, and 0·3% on the Continent. Thus it is possible that these three children were infected directly through the air passages from subjects with "open" tuberculous lesions in the lungs due to bovine strains. Further proof that the primary lung lesions did not have a hæmatogenous origin was obtained from a small series of cases of

tuberculosis of bones and joints occurring in children in the surgical wards of the hospital, from whom 20 strains were isolated from material obtained from the diseased bones or joints. In six other cases which came to post-mortem, bone lesions were found, but were secondary to abdominal or thoracic disease, and in these cases the infecting bacilli were only isolated from the primary lesions. Of the total 26 strains thus obtained, nine (34·6%) were found to be bovine strains. Tuberculous disease of bones and joints is nearly always due to a blood-borne infection, and the percentage of bovine strains which we found in such cases was more than ten times higher than in cases with primary thoracic tuberculosis.

Most of the photographs in this article are the work of Mr. John Kirkpatrick of the Pathology Department at the University of Glasgow. I would like to express my appreciation for the care which he has taken with the work.

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JOINT DISCUSSION No. 2.

Section of Neurology and Section of Surgery.

Chairman—Mr. C. H. FAGGE, M.S. (President of the Section of Surgery).

(*December* 2, 1931.)

DISCUSSION ON THE DIAGNOSIS AND TREATMENT OF ACUTE HEAD INJURIES.

Dr. George Riddoch: The subject for discussion is opportune, not only because the frequency of head injuries from accidents appears to be on the increase, but also because there is no doubt but that efficient treatment in the early stages does much to prevent many of the sequelæ. Some of the methods of treatment still in vogue are either wrong or inadequate, and could be corrected with beneficial results.

Diagnosis of acute head injuries.—Everyone will now agree that the presence or absence of fracture of the skull is, in general, unimportant compared with that of intracranial injury. Extensive fractures, especially of the base, and depressed fractures are, as a rule, associated with damage to the brain, with or without hæmorrhage, and compound fractures introduce the risk of sepsis. But severe cerebral injuries may occur without fracture of the skull. In a series of 441 cases of head injury McClure and Crawford [4] found that 171, or 39%, were unassociated with fracture.

Contusion is by far the most common and important traumatic lesion of the brain, and I propose to confine my remarks mainly to its more immediate clinical manifestations, after first discussing the phenomena of concussion.

Concussion.—Trotter [8], to whom all will pay tribute for his pioneer work in this field, as in other fields of medicine, has defined concussion as "a condition of widespread paralysis of the functions of the brain, which comes on as the immediate consequence of a blow on the head, has a strong tendency to spontaneous recovery, and is not necessarily associated with any gross organic change in the brain substance." Many theories have been advanced to explain the causation of the symptoms, but that of acute cerebral anæmia, the result of sudden deformation of the skull, so ably advocated by Trotter, is probably correct.

The symptoms are immediate on receipt of the blow, the disturbance of function varying in severity and duration. In slight cases there is merely transitory dizziness or loss of consciousness followed by mental confusion and physical weakness which last for a short time. The pulse and respiration rate are usually unaffected. The patient may then recover completely, or, for a few hours, complain of headache. In such a case, concussion is limited to the cerebrum. Sometimes, after relatively mild concussion, complicated activities, such as playing football, are carried out without the patient afterwards remembering anything of them. Behaviour of this sort, however, has always the stamp of imperfect conscious guidance, for it is obviously automatic and poorly adapted to the changing circumstances of the game.

The clinical picture of severe but uncomplicated concussion is said to be as follows: The patient lies limp in the position in which he has fallen. He is pale, his skin soon cools, and if there is an open wound it does not bleed. His pupils are dilated and may be fixed, his pulse may be slow, or if the condition is more serious, rapid and feeble, or even imperceptible. His blood and cerebro-spinal fluid pressures are low. Breathing for a short time may stop, and when it reappears it is shallow, with periodic sighing. All reflexes may be abolished and the patient fails to respond to stimulation. Such a clinical picture indicates complete loss of the functions of the cerebrum and upper part of the brain stem, with severe impairment of bulbar function. Of the bulbar centres, the respiratory

is the first to be put out of action, but if the heart continues to beat and breathing quickly starts again recovery is the rule. If breathing is arrested, artificial respiration may keep the patient alive until the respiratory centre recovers again. Death, however, may occur, presumably from bulbar anæmia. It is said that at autopsy no demonstrable lesion may be found, but as a rule there are contusions of the brain-stem. In Vance's [9] series of 507 post-mortem examinations in cases of head injury, death in concussion occurred in 139, or 27·4%, and of these 86 patients died within a few minutes. A blow of moderate strength in the suboccipital region is more likely to result in concussion of the medulla than a similar blow on the vault. In all but the severe cases, however, recovery from this state of collapse commences within a few minutes. The heart begins to beat more strongly, respirations, although irregular, become deeper, reflex reactions return, the pupillary response to light being the first, the degree of unconsciousness becomes less and the patient begins to stir. He usually vomits, and the straining, by raising the blood-pressure, hastens recovery. During this period, or "stage of reaction" (Trotter) [8], there may be restlessness, irritability and sometimes mild delirium, and headache is constant. The temperature may be slightly raised.

With a history of head injury the recognition of concussion is not difficult. As Trotter [8] has insisted, the three essential clinical features of it are (1) the absolutely instantaneous onset, (2) the paralytic nature of the symptoms, and (3) their tendency to disappear spontaneously. Difficulty, in the acute stage, arises when complications, such as contusion, laceration and hæmorrhage, are associated with it. It is clear that the brain has been damaged or extracerebral hæmorrhage has occurred, if recovery of unconsciousness is delayed, if the stage of reaction is prolonged more than a day or so, if stupor or coma recurs after consciousness has been regained partially or completely, if local paralysis is present, or if blood is found in the cerebrospinal fluid. But experience of the subsequent histories of patients who have survived concussion must compel clinicians to go further, and to doubt whether any but the mild or moderately severe cases recover without organic damage to the brain. Even these do not always escape.

In regard to the duration of unconsciousness in concussion, the condition of the cardio-vascular system may play a part, for if the heart is feeble and the arteries are diseased, re-establishment of the circulation may be delayed and the period of unconsciousness be thus prolonged. Extracranial hæmorrhage would have the same effect, and must be taken into account.

Contusion and laceration.—The essential difference between contusion and laceration, which are by far the most common structural lesions of the brain after head injury, is in the degree of hæmorrhage. In both there is focal necrosis of neural elements, but in laceration there is often considerable bleeding from torn vessels, whilst in contusion, hæmorrhage, as a rule, is little more than punctate.

Local cerebral contusion, followed by persistent paroxysmal headache, may result from a blow on the vault that gives rise to only mild concussion, in which the patient is merely dazed for a brief period. On the other hand, it is probable that severe concussion, especially when its severity is indicated by the presence of fixed pupils and bulbar signs, is almost invariably associated with multiple contusions throughout the brain. Apart from pathological evidence, this view is supported by the frequency with which symptoms such as certain kinds of headache, giddiness, impairment of memory and so on, which are not psychogenic, follow these injuries, and become chronic.

The presence of multiple contusions results in prolongation of the period of unconsciousness in concussion, and recovery from it is protracted, due probably to œdema round the areas of contusion. The clinical picture has for long been ascribed to "cerebral irritation," but whilst there may indeed be increased excitability

of the nervous elements owing to venous congestion and anoxæmia, defective inhibition of neural function plays a part, for at least many of its symptoms are of this order. The symptomatology, at all events, is well known, and need only be briefly described. After the initial phase of collapse, which may last several minutes or hours, the patient more or less regains consciousness but is irritable and drowsy during the day, and is often delirious or may even be maniacal at night. He resents interference, lies curled up in bed with his eyes turned away from the light, and, especially if disturbed, complains of severe throbbing headache. There may be nausea and vomiting, the temperature is often slightly raised, and the pulse full and bounding. This condition usually reaches its height within about two days of the injury and may persist unabated for days or longer. After this, as a rule, most of the more acute symptoms such as fever, delirium and photophobia, disappear, but an abnormal state of health often persists for an indefinite period of time.

The most common symptoms of cerebral contusion, which were looked upon as neurotic before Trotter indicated their organic character, are headache, giddiness, irritability, nervousness, defective memory and power of concentration, fatigability, lack of interest and initiative, and sleeplessness. The headaches, which in the early days are continuous, later become paroxysmal and from their clinical features can be differentiated into two classes.

(1) *Intracranial hypertension headache.*—This is the more common. Briefly, it is a severe aching or bursting pain with additional throbbing or shooting pains, which are brought on by emotional disturbance, bright light, and effort such as reading, listening intently, hurrying, straining at stool, coughing or sneezing. The headache is also aggravated or precipitated when the patient lies down for any length of time, especially if asleep, as in bed at night, and is relieved when he sits up and keeps quiet.

Such headaches, in the acute and subacute stages after head injury, are probably due to cerebral œdema. It is unlikely that œdema persists for long, and some other pathological abnormality is present, possibly damage to the absorbing mechanism of the cerebrospinal fluid from lesions of the Pacchionian bodies. Confirmatory evidence of increased intracranial tension is usually supplied by abnormally high manometric readings of cerebrospinal fluid pressure at lumbar puncture.

(2) *Intracranial hypotension headaches.*—In a much smaller number of cases the headaches, at all events when they become intermittent, react quite differently to posture, for they diminish or disappear when the patient lies flat, and reappear when he sits up. In such cases the cerebrospinal fluid pressure is abnormally low. McCreery and Berry [5] found diminished pressure in 12 out of 414 cases. A characteristic example is the following case :—

Cerebral contusion with intracranial hypotension headaches.—C. N., aged 32, steel erector. Two years ago he was hit in the middle of the forehead by the piston of a compressed air gun, and was unconscious for about an hour. A scalp wound was stitched up and he remained at home for a week but did not stay in bed. Following the accident he suffered from continual stabbing pains in the region of the wound, which were worse on coughing and stooping, and were apt to keep him awake at night. Three weeks after the accident, in addition to the pain in the region of the wound, he began to get continuous throbbing headaches in the temples and top of his head, which were worse in the evening and were aggravated by coughing and stooping. The throbbing was synchronous with his pulse, and with the headaches he had attacks of giddiness.

On examination, there was weakness of both external recti, with diplopia on looking to either side, but his nervous system was otherwise clear. His cerebrospinal fluid pressure was however found to be subnormal, being 80 mm. of cerebrospinal fluid. His headaches, apart from the stabbing pains in his forehead, disappeared when the foot of the bed was elevated.

The cause of the lowered intracranial pressure in such cases of post-contusional hypotension headaches has not been proven, but it may be that the choroid plexuses are damaged, with consequent diminished rate of production of cerebrospinal fluid.

Rand [7], from histological investigation in fatal cases of head injury, has demonstrated the frequency of gross lesions of the choroid plexuses.

The late clinical results of cerebral contusion do not form part of this discussion, but some of the sequelæ must be touched upon in so far as they indicate the organic basis of many of the symptoms. In the vast majority of cases in which recovery is incomplete after the stage of concussion, the symptoms complained of, and for which contusion is probably the main organic foundation, are headache, giddiness and minor mental disorders such as defective memory and concentration, slowness of thought, loss of initiative, insomnia and irritability. Engrafted on this state of mental enfeeblement, and as the result of anxiety from different causes, other symptoms may develop.

Gross mental defect, sufficient to constitute insanity, for longer than a few days after the injury, is uncommon, but such cases do occur, and two which have recently come under observation may be briefly described.

Traumatic delirium.—G. B., a man aged 63, who had led an exceptionally healthy and active life, was thrown from his horse on to his head. Unconsciousness was immediate, but five minutes later he had recovered sufficiently to ask for his wife, and within ten minutes recognized her. He bled for three days from his left ear and nostril. Soon after the accident he vomited repeatedly and was incontinent. There were no convulsions. He passed into a state in which, when not under the influence of sedatives, he was restless, violent, disoriented and confused. Usually he recognized his wife, but no one else, and he never knew where he was. He complained of severe headache. For the first four weeks he took food well, but after that would take fluids only.

Five weeks after the injury, when I first examined him, he presented no sign of focal damage to the brain. The cerebrospinal fluid was clear (and was subsequently found to be normal), but appeared to be under pressure. For lumbar puncture a general anæsthetic was necessary because of his violence.

Since the restless, confused mental state was punctuated by three stuporose periods, and the cerebrospinal fluid pressure appeared to be high, the possibility of subdural venous hæmorrhage as a complication of multiple cerebral contusions was considered. After the journey to London, reflex changes in the left lower limb were for the first time present. The left knee- and ankle-jerks were relatively increased and the plantar response was extensor. Cairns tapped the anterior horns of the ventricles, which were found to be full of cerebrospinal fluid, but the pressure was subnormal, being 90 mm. of cerebrospinal fluid. The arachnoid was distended with cerebrospinal fluid, and bulged through the openings in the skull. These observations clearly indicated that there was no compression of the hemispheres. The patient's condition after the operation remained the same as before until he developed pneumonia a week later and soon afterwards died.

At post-mortem examination the brain appeared to be shrunken, and there were superficial lacerations on the outer aspects of both temporal lobes. Gross hæmorrhage was not found. Sections of the cerebrum, brain-stem or cerebellum did not show, to the naked eye, any obvious abnormality. Fixation is not complete, and histological examination has yet to be carried out.

The abnormal mental state in this case, which persisted for seven weeks, till the patient died from pneumonia, was one of delirium, with periods of maniacal excitement or, more rarely, of stupor, the pathological basis of which cannot at present be explained. It cannot have been due to venous congestion from cerebral œdema throughout the whole course of his illness, for although the evidence in regard to cerebrospinal fluid pressure was equivocal, the brain at operation and at post-mortem was, if anything, shrunken, and the arachnoid was distended with fluid. Presumably multiple contusions will be found.

The second case is also instructive, for it illustrates the rare condition of severe traumatic dementia.

Traumatic dementia.—Private E. T., aged 28, Army clerk, on March 13, 1931, was admitted to Cambridge Military Hospital unconscious, and was said to have been run into by a motor cycle. He was unconscious and incontinent for eleven days, after which time he

was restless and irritable, but was able to indicate when he wanted the bed-pan. There was a contused wound in the occipital region, and skiagrams showed a vertical fracture extending down the whole length of the occipital bone into the foramen magnum. Nine days after the accident unconsciousness deepened, and on lumbar puncture the cerebrospinal fluid was found to be under increased pressure, to be yellow and to contain blood. Thirteen days after the accident he answered questions sensibly, and no organic signs were found in his nervous system. He was kept flat in bed for nine weeks, and in hospital for fourteen weeks. On discharge he seemed to be fairly normal, except for paroxysmal headaches, forgetfulness and insomnia. He had been a particularly competent clerk, but when he returned to duty he was found to be quite useless. He did not know his work, was grossly forgetful, slow and stupid, and lacked initiative. He was invalided from the Army in September, 1931.

He was taken to see me on November 5, and was still suffering from headaches. He was dull and slow mentally, and his memory was very poor. According to his father, he had, since the accident, completely altered in character. Previously an intelligent, thoughtful and affectionate man, he had become simple, talkative and argumentative in a silly way. He was mischievous, and would break up things, for example a bicycle, in a way that a child would do. Apart from anosmia and diminished hearing, there were no abnormal physical signs.

Although at first sight this picture of mental deterioration is very different from the minor mental changes common to slighter cerebral injuries, the difference is, on reflection, only one of degree.

Focal signs with cerebral contusion.—It is a matter of common medical experience, but to the legal mind difficult to believe, that traumatic cerebral injuries which give rise to disabling symptoms of organic origin are, as a rule, unaccompanied by demonstrable physical signs. This difficulty in comprehension encountered in the Law Courts is of the same order as that in regard to the significance of fracture of the skull. Still, physical signs are more common than is generally supposed, but they may often be missed, for most frequently they are either pupillary, and will escape detection unless the examination is careful, or consist of defects of smell or hearing, functions which may not be tested unless the patient draws attention to them. Occasionally slight cerebral signs are present in the form of evidence of pyramidal defect or sensory impairment. Pupillary abnormalities are usually due to lesions in the mid-brain, and consist of irregularity, inequality, eccentricity, and impairment of reflex reactions. Other ocular signs are sometimes encountered, such as diplopia, squint, and weakness of conjugate movement of the eyes. When the defect is for conjugate lateral movement, the lesion, of course, is in the pons.

In my experience, mesencephalic signs in the early stages of severe head injuries are not uncommon, but many of them rapidly disappear. A recent case may be cited.

Contusion of mid-brain.—J. B., aged 25, female. On November 9, 1931, she was apparently thrown from her horse, and was found lying on the road unconscious by a doctor, who said that she had a series of generalized epileptic attacks. At that time her left pupil was larger than her right, and she vomited. There was a bruise in her left occipital region and an abrasion in her left frontal region. No bleeding from her ears or nose was noticed. She was taken to hospital, where her pulse was found to be 56, respirations 12 and temperature subnormal. In three days she slowly regained consciousness, but when I saw her on November 13, 1931, four days after the accident, she was still dull and confused, but had no more fits or vomiting. On that day her pulse was 64, respirations 16, temperature 99·4. She objected to light, complained of bilateral frontal headache, and was able to co-operate, although her answers were slow. Her discs were clear, her pupils moderately dilated and reacted fairly well to light but poorly on convergence. There were absence of conjugate convergence with slight divergent squint, and diplopia on upward and downward movements of her eyes, due to weakness of the elevator and depressor of her right eye. Coarse lateral nystagmus on looking to either side was present. There was slight weakness of her left lower face and complete deafness to a watch in her left ear. All voluntary movements of her limbs were good and equal. Her left supinator jerk was

brisker than her right, her abdominal reflexes, as well as her knee- and ankle-jerks were brisk and equal on the two sides, and her plantar responses were extensor. Sensibility to light touch and pin-prick appeared to be good. Her heart was clear, and blood-pressure readings were 120/70. There had been retention of urine and fæces since the accident. She was not lumbar punctured. Up to that time she had been kept flat in bed, but was now to be propped up for part of the day and given a daily rectal injection of three ounces of magnesium sulphate in six ounces of water.

On November 24, 1931, fifteen days after the accident, her doctor reported that she had remained mentally clear, but continued to complain of some headache. She was sleeping well with the help of a moderate dose of bromide and chloral. Diplopia and squint had disappeared, but there was still nystagmus, but now only on looking to the left, and her plantar responses were indefinite.

The physical signs in this case were presumably due to contusion of the mid-brain, and it is likely that few of them will be permanent.[1]

The occurrence of convulsions in the stage of concussion, as in this case, is no indication that the patient will be subject to fits. As is well known, less than 10% of the soldiers who were wounded in the head in the war suffered from late recurrent epileptiform seizures, although a much larger number had fits soon after the injury. Nevertheless, it is to be remembered that such attacks may develop for the first time months or even a few years after an accident to the head, to which they must, in all probability, be attributed, even if the cerebral damage may possibly act only as a precipitating cause in potentially epileptic subjects.

An interesting and rare syndrome occurring in association with ocular abnormalities is Parkinsonism, of which I have in my records three examples. Apart from their history of headaches and giddiness, they closely resemble, in their ocular and Parkinsonian features, cases following encephalitis lethargica. None, as yet, have been reported in this country, but clinicians on the Continent are familiar with them.

Treatment.—Head injuries, almost more than any other group of cases, justify the establishment of neurological departments in the large general hospitals, so that the neurological physician and surgeon can combine in tackling the therapeutic problems which are as yet imperfectly solved. It can, however, be said at once that apart from the necessity for dealing with wounds and the comparatively rare complication of compressive hæmorrhage, surgery plays little part in early treatment. Many questions in treatment as in other aspects of the problem require discussion, and attention will first be directed to the common case of slight concussion, in which the patient, for a brief period, is dazed or even unconscious. Usually recovery is complete and permanent, but in view of the possible subsequent development of so-called contusional headaches, should rest in bed without or with active treatment be insisted upon, and for what period of time? Ideally, rest for a week or ten days is advisable, but practically, this may not always be possible, for the circumstances of the accident and the psychology of the patient demand consideration.

When unconsciousness is, however, of longer duration the patient ought to be put to bed in the recumbent position, and, if collapsed, external warmth applied.

1 POSTSCRIPT.—Since this paper was read, I examined this patient again, on December 5, 1931, twenty-six days after the accident. She did not remember the previous examination or having seen me before. Headaches, which she described as a sensation of pressure on the top of her head, made worse by sitting up, had steadily diminished in frequency and severity. Giddiness was also less. There had been no diplopia for some days, and she was sleeping well. She knew that she was irritable.

On examination, she was clear mentally and co-operative. The positive signs found were as follows. Her pupils were equal and moderately dilated, but irregular, and the right reacted very poorly to light but the reaction on convergence was good in both. There was still some lateral nystagmus. Sensibility to light touch and pin-prick was slightly diminished on the left side of her face, and hearing on that side was still impaired, a watch being heard at four inches. The tendon jerks in her limbs were equal but diminished, as were also her abdominal reflexes, and her left plantar response was still extensor although her right was now flexor. There was some reduction in sensibility to light touch and pin-prick in her left upper limb, but postural sensibility was good. On lumbar puncture, the cerebrospinal fluid was found to be blood-stained, and under a pressure of 110 mm. to 120 mm. of cerebrospinal fluid.

If collapse is severe, the lower end of the bed may be raised, or the lower limbs bandaged. Pituitrin is said to be useful, but I have no personal experience upon which to give an opinion. For respiratory embarrassment, a hypodermic injection of strychnine can be given. Apart from attending to external wounds and keeping a watch on the pulse and respiration, the patient at this stage should be disturbed as little as possible. After the stage of reaction has clearly developed and more or less complete consciousness has returned, it is probably wise, as a routine, to perform lumbar puncture in order to estimate manometrically the cerebrospinal fluid pressure, and to ascertain whether the fluid is mixed with blood. If the pressure is high—the normal limit being 200 mm. of cerebrospinal fluid—the patient should be propped up, so using gravity as a means of lowering the intracranial pressure; if on the other hand, it is subnormal—below 100 mm., as it is in a smaller number of cases—the recumbent position is to be maintained. Should the pressure be within normal limits, the safe plan is moderately to raise the head and shoulders, say with two pillows.

In regard to the presence of blood in the cerebrospinal fluid, if it is so excessive as to indicate continued hæmorrhage, no appreciable amount of fluid should be withdrawn. But if the quantity of blood is such that it is freely mixed with the cerebrospinal fluid, the question arises as to the advisability of immediate drainage, in order to diminish the development of aseptic meningitis. The inflammatory reaction to blood in the subarachnoid space is now well recognized. Its clinical effects are best shown in cases of non-traumatic subarachnoid hæmorrhage from rupture of a basal aneurysm. One of the questions to be answered by investigation is whether meningitis from hæmorrhage in the acute stages of head injury endangers life, as, for example, by interfering with the functions of the bulb or its nerves, or gives rise to some of the sequelæ of head injury.

Withdrawal of fluid by lumbar puncture is also used as a method for lowering intracranial pressure when it is unduly high, and has to be considered on this basis.

Methods of lowering intracranial hypertension.—There are, apart from utilizing the posture of the patient, three methods in present use for reduction of raised intracranial pressure: (1) drainage by lumbar puncture, (2) dehydration by hypertonic solutions, and (3) decompression.

(1) Lumbar puncture.—Since 1905, when Quincke first advocated it, drainage by lumbar puncture has had supporters and detractors. Cushing advised caution. Jackson [2] is in favour of its being employed at intervals of from six to twenty-four hours. Ochsner [6] advocates the following procedure. If the cerebrospinal fluid pressure is above 10 mm. Hg, it is abnormal, and enough fluid should be slowly removed to decrease the pressure above 10 mm. by one half—thus, if the pressure is 20 mm. Hg, to reduce it to 15 mm. Hg. My feeling at present is that its usefulness is better restricted to the removal of free blood, dehydration being probably safer.

(2) Dehydration.—Hypertonic solutions of salt, magnesium sulphate, and glucose are employed. The first and last are given intravenously, but magnesium sulphate ought to be administered only by mouth or rectum, for in the blood it acts as an anæsthetic and depresses respiration. For intravenous use, a 50% solution of glucose has advantages over salt in that it dehydrates more gradually and does not lead to a subsequent rise of pressure. Fifty to one hundred c.c. are injected once or twice daily or even more often.

Provided that the patient can retain it for fifteen or twenty minutes, an injection per rectum of 6 oz. of a 50% solution of magnesium sulphate gives excellent although less rapid results. It should not be repeated more than twice daily, because of the risk of irritation of the rectal mucosa. Should this arise, a weaker solution, for example, 1½ oz. of magnesium sulphate in 8 oz. of water, can be used. Fay [1] has

proved that this weaker solution has a pronounced effect within an hour of injection. He has also shown that magnesium sulphate is twice as effective a dehydrating agent as sodium chloride, when introduced into the intestine.

During dehydration, the intake of fluid ought, of course, to be limited. Abstraction of fluid from the body, if pushed too far, is dangerous, but employed with moderation it is a most valuable method for reducing intracranial pressure.

(3) Decompression.—Except in cases of compression from hæmorrhage, this procedure has been superseded by dehydration and lumbar puncture drainage.

There remains the question of how to deal with those cases in which intracranial pressure is abnormally low. Is there any need for treatment, in addition to rest? It is known that sequelæ such as headache of the hypotension variety do occur and persist indefinitely. Can they be prevented by active measures in the subacute stage of the illness? Some believe that they can. As a means of raising the intracranial pressure towards the normal level it is at least reasonable to keep the patient in the recumbent position, or even to prop up the foot of his bed. Leriche [3] has advised intravenous injection of hypotonic solution or sterile water, and McCreery and Berry [5] claim good results from their use.

In regard to sedatives, which are often necessary in the treatment of head injuries in the early stages at least, morphia should not be employed because of its depressant effect on the respiratory centre. Bromide and choral are safe.

The minimum period of time during which complete rest in bed should be insisted upon will, of course, vary with the severity of the case. I would suggest that three weeks is necessary in cases of moderately severe concussion, in which there is reason to believe that some contusion is present. After this time, if symptoms have gone, the patient should be allowed to get up for increasing periods, but should headache or giddiness return, he should be put back to bed for another week or ten days. During convalescence, which should last for another three or four weeks, his activities should be carefully regulated. Lastly, an atmosphere of anxiety should be avoided, for after head injury, neuroses are apt to sprout with disconcerting ease.

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Mr. Geoffrey Jefferson: In spite of all that has been written and said on the subject of head injuries, it must be admitted that only now are we commencing to reach any real unanimity of thought and practice in treatment.

During the past hundred and fifty years surgeons exercised all their ingenuity and used all their practical skill (and both were considerable) in attempting to solve the problem of the fractured skull, in inquiring as to why the fracture line followed such and such a course, why it varied so much in different cases, why it appeared so frequently in places which clearly could have received no direct contusion. From the time of Pott onwards, a number of valuable studies were made, culminating in the wide-spread acceptance of the theory of bursting which comprehends, but does not explain, all the varieties of bone injury. The question of the fracture itself is, however, unpopular to-day, and surgical teaching generally passes over this aspect of head injury without deeply concerning itself with it. This is a matter of policy rather than a lack of lively interest, because it has been necessary to instil into the minds of students and others the fact that it is the intracranial damage rather than the cranial which is of vital importance. The fascinating study of the mechanics of bone injury, which we are better able to attempt to-day than ever before, has been for this reason suspended. I shall have little concern with the bony damage on this

occasion and although depressed fractures have their interest and importance I shall leave comment on them on one side and pass on to the consideration of the acute intracranial injury.

At the outset I must make a declaration of my beliefs which are these:—

(1) That in all head injuries serious enough to call for treatment during the acute stage, there is a fundamental state due to general cerebral contusion and that this has a recognizable histological picture, a state in which compression may be, but often is not, present according to circumstances, though secondary œdema may develop later.

(2) That there are laid down on top of this certain "Epiphenomena", of which the chief are the various gross hæmorrhages, intra- and extra-dural, and those other lesions specific to the fracture alone—meningitis, intracranial aerocoele, and some of the cranial nerve palsies.

(3) Lastly, that any patient who on admission to hospital is unconscious, may safely be assumed to have definitely widespread cerebral neural damage of greater or less degree. In addition, he may have any of the epiphenomena and it is a fascinating clinical task to separate the signs of these off from the fundamental state of general cerebral contusion.

The intracranial injury.—A discussion of the physical conditions which underlie that clinical picture of stupor, restlessness, and resentment with which everyone is familiar, leads us quickly into a thicket of old-established terms, notably concussion, contusion, and compression, each with its list of traditional signs. My own inclination is to dispense with concussion altogether and to retain two terms only—contusion and compression, for with these two and these two alone are we on firm ground. The defect of the word "concussion" is not that it is meaningless, but that it suggests too many unproven possibilities. Before we can profitably discuss this matter it is necessary that we should clear the way by a restatement of the views accepted to-day on the meaning of these states and to review Trotter's original conception.

Concussion.—Concussion, as Trotter conceives it, is a momentary affair during which the cerebral circulation is arrested by the instantaneous compression of the intracranial contents as a whole by the injuring force, a force generally of great mass but relatively low velocity. It is clear that the duration of such vascular arrest is to be measured in seconds, or less, and recovery should be extremely rapid. If, on the other hand, the unconsciousness and disablement last longer than a few seconds or minutes, Trotter postulates cerebral damage, and regards the stuporous state of the patient as due to "cerebral contusion" and not concussion. It follows therefore that any patient who is unconscious for more than a little time after the accident, and who is in fact unconscious on admission to hospital, must be suffering from an injury more severe than the effect of a temporary circulatory arrest. This conception, although simplicity itself, very quickly involves us in difficulties, chiefly through our inability to set a period to the duration of concussion, and to know with exactitude when it merges into contusion.

Trotter's conception of circulatory arrest as the cause of concussion is attractive and has been readily received by neurologists because he invokes a primarily non-neural mechanism to explain it. Very possibly time may prove it to be an incomplete or even incorrect explanation, as Miller recently suggested, but with our present knowledge it is the only one which easily explains rapid recovery from a transient stupor. It is perhaps difficult for us to imagine a force which is severe enough to arrest the circulation and yet causes no change whatever in the cerebral mass. We cannot criticize the idea too strongly because we are unable to set anything else with certainty in its place. We do not know enough of the possibilities of a trauma just severe enough to cause temporary unconsciousness any more than we know with certainty why it is that a contusion may interfere temporarily with conduction in a peripheral nerve. It may be a circulatory effect or it may be something more subtle and elusive—electrolytic and hydrogen-ion concentration variations and the like—as Rahm, and Knauer and Enderlen have suggested. The practical point is that we do

well to accept concussion, whatever its actual pathology may be, as a dogma, as the symbol of a state we know to exist and of which primary shock and retrograde amnesia are the only immediate specific results.

This is in the end a sterile subject, and a futile one, for the pursuit of theory may well prejudice the welfare of our patients. It is as clear as anything can be that a most precise knowledge of the cell changes underlying the state of pure concussion, even if we could verify them in the human subject, would not be of any real clinical assistance to us. The one great lesson that is of service is that which teaches that patients who remain stuporous after a head injury have suffered a contusion of the brain. What the nature of that contusion may be is a fitting subject for further remark.

General cerebral contusion.—The term cerebral contusion also requires a certain amount of critical consideration. In the first place the word contusion calls up to the mind a local injury, and although a local injury of the brain may be, and often is, present, this is not quite what the phrase ought to imply. We really mean a general contusion of the whole brain, for although chance may have ordered matters in such a manner that there has been damage done to the brain at the point of impact, yet by the laws of hydrodynamics every part of the intracranial contents must have suffered equal damage, subject to such modulations as the dural septa and different tissue resistances may bring about. Further, the term contusion implies hæmorrhage. It is a commonplace of observation on post-mortem material that small hæmorrhages may be present, scattered through the cerebral tissues, some in the white matter, others in the grey, or at the junction of the two (see fig. 1). And we may assume that these punctate hæmorrhages, verified at necropsy, also occur in some degree in patients who recover. It requires statement that these hæmorrhages are not so widespread as popular medical fancy has pictured. They are, in fact, usually quite limited in their distribution, notably to the poles of the brain and in the immediate vicinity of any laceration which may be present. Thus a section at necropsy, an inch or two from the tip of the frontal lobe, may show many punctate hæmorrhages, whilst a transverse section through the motor areas or further back may in the same subject reveal none whatever. This calls to mind the classical dispute between Kocher and von Bergmann on the true pathological nature of concussion.

Histological examination of these punctate or larger hæmorrhages shows the damage to projection and association tracts which we should expect to find (fig. 2). In the neighbourhood we find the perivascular sheaths infiltrated with blood, some tight-packed, others loosely spattered with erythrocytes. Whether the blood in and around these sheaths has drifted in from the surface, or has come from the intracerebral arterioles, we can quickly decide. It is clear that the red cells are too tightly packed to have flowed in but have arisen from rupture of the vessel wall. We must remember that when the head is deformed by trauma not only is the brain substance itself subjected to violence but also the blood within it. The violence may be great enough to cause the vessels to burst within the brain, and we may assume that the blood in the sheaths has come from the vessels within and not from the surface. It is true that these hæmorrhages may occur not only in the hemispheres but also in the brain-stem and medulla, as Duret in particular demonstrated. But these hæmorrhages are usually very sparse and it may be questioned whether they are themselves the cause of death rather than the evidences of a dynamic force which has acted on vital structures in other ways as well.

Polar contusion.—It has long been known that the frontal and temporal lobes may show considerable laceration during the momentary violent deformation of the skull that injury produces. The fact that these lacerations tend to affect the poles of the frontal, temporal, and more rarely the occipital lobes seems to me to render the term polar contusion a singularly fitting appellation. It is certain that injuries of these lobes may lead to quite definite neurological signs of localizing import.

I need only mention anosmia and incontinence continuing after consciousness has been regained, as signs of frontal contusion, and visual field defects (of which I have seen three examples) as evidences of focal injury. These polar contusions, in

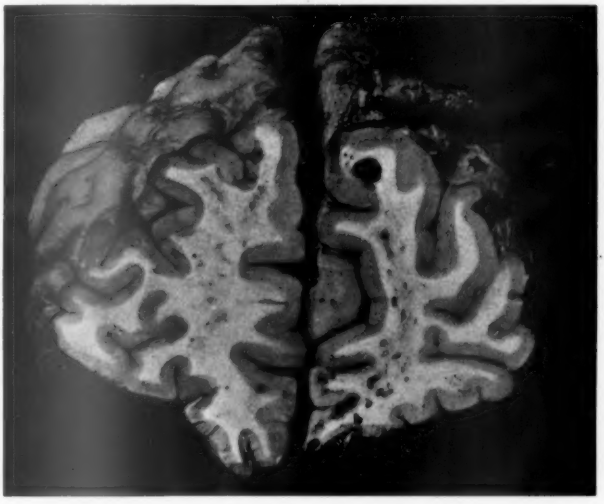

FIG. 1.—Coronal section: Frontal lobe showing punctate hæmorrhages.

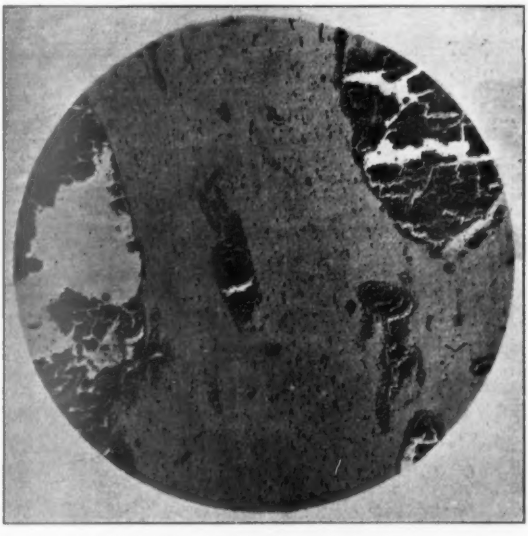

FIG. 2.—Hæmorrhage into the white matter.

which the leptomeninges and cortex are often severely damaged, lead to bleeding, often slight, but at other times resulting in the production of an acute subdural hæmorrhage large enough to cause a rapid fatality.

FIG. 3.—A perivascular space packed with blood.

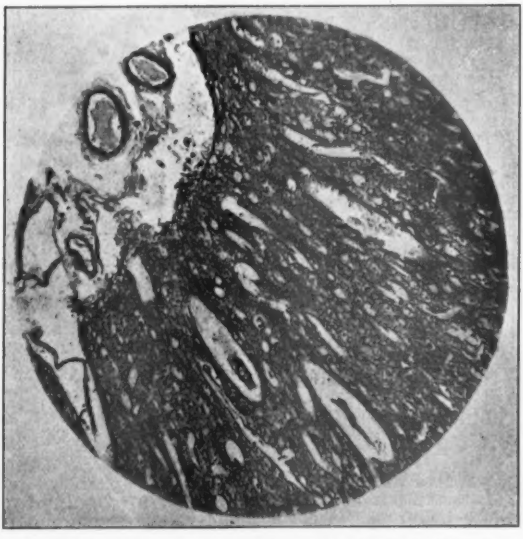

FIG. 4.—Section from bottom of a sulcus; radiating œdema cerebri, three days after injury.

The factor of the cerebrospinal fluid in cranial trauma.—We owe to Duret the conception that the cerebrospinal fluid plays a large part in the pathology of head injuries, although he may have exaggerated its importance. The outstanding histological picture in cases which survive to the second or third day is œdema. This is evident in the illustrations which follow, in the dilatation of perivascular sheaths and in the actual pericellular œdema seen very well in fig. 5. The question whether these changes are the artefacts of fixation and staining will come to mind. But against this is the fact that frozen sections show much the same conformation. The work of Henschen, of Le Count and Apfelbach, and of Rand, also agrees that œdema is the most constant of post-mortem findings. Apfelbach discovered gross increase in weight and diminution in ventricular size, probably the two most convincing arguments that could be brought forward. Latterly Rand has turned his attention to the choroid plexuses, and finds often a waterlogged stroma with definite changes in the cells, leading presumably in some cases to over-filtration of

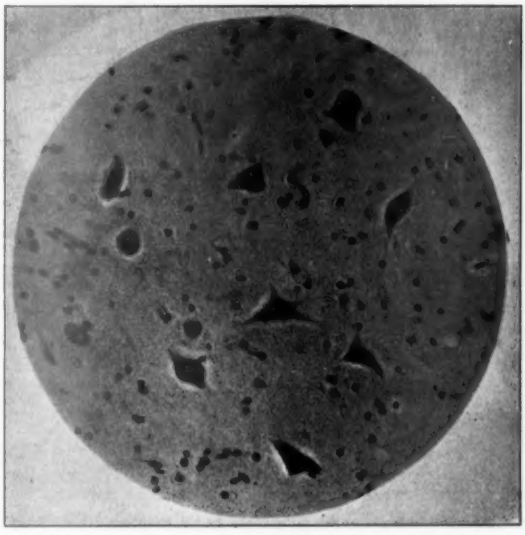

FIG. 5.—Pericellular œdema, thirty-six hours after injury.

cerebrospinal fluid, in others to under-production. These factors, together with the blocking of arachnoid tufts by blood-corpuscles, no doubt account for the fact that both "wet" and "dry" brains may be found after injury, and that in some the brain is large and bulging, while in others its size is not noticeably altered. The difficulty that I find myself in is to know with certainty how much of this œdema is early and how much of it is late. It is quite clear that the cerebrospinal fluid must be subjected to the same trauma as the other intracranial contents, and may equally be displaced. We may, therefore, be faced, in generalized cerebral contusion, with neural disruptions produced by the cerebrospinal fluid. It is my belief that this is so, and that in this fact, together with reactionary œdema, we have the solution of the ancient conflict of opinion between Kocher and von Bergmann. Vascular hæmorrhages are obvious to the naked eye; cerebrospinal fluid "hæmorrhages" can only be discovered by special study. (Dr. Greenfield is of the opinion

that both the blood and some of the œdema are outside the lining of the Virchow-Robin spaces. This is likely to be true as these spaces are not known to be so easily distended. The blood in particular no doubt injects itself along the vessels after rupture in a pathway of least resistance.)

From the foregoing we have one at least of the methods by which the cerebro-spinal fluid comes to play a part in head injury. In addition, the intraventricular fluid may be of importance by producing, as it were, an internal pressure cone suddenly acting on the medulla and, maybe, tearing it, as Duret conceived. Very little evidence exists on this point, and I have looked for it without finding it once. It is possible that hyperthermia is due to changes of this nature in third ventricle vegetative nuclei rather than because of hæmorrhages into the pons. In addition, the fluid in the basal cisternæ may be compressed, and these might give way, thus releasing those collections of subdural and extra-arachnoidal fluid that Naffziger and

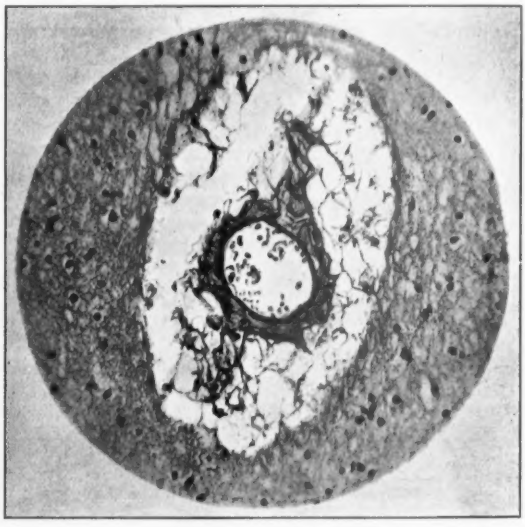

FIG. 6.—A perivascular space showing œdema and disruption, three days after injury.

most of us have observed. The extra-arachnoidal fluid is something of a conundrum, for we know so little of what alters the permeability of the arachnoid, though it is likely to be a colloidal change in the fluid rather than a reversal of the membrane powers.

The common necropsy picture of cerebral œdema brings us up sharply against the question: is compression an important factor in bringing about the unconsciousness and stupor in which we find these patients for hours or days—or even weeks—after an injury? The general opinion seems to be that if the patient does not recover consciousness within a few hours of the injury, his state is due to cerebral compression, possibly due to gross hæmorrhage, perhaps due to general brain swelling. Now although from the practical point of view it is wise and proper that we should regard our patients from this angle, it is a fact that a number of these patients are not actually suffering from compression at all. This conclusion is arrived at from three distinct observations which others may apply for themselves.

First, that the cerebrospinal fluid pressure is often low or not notably increased, as measured by lumbar pressure readings. Second, that the effects of intravenous administration of hypertonic salt or glucose solutions are not uniformly good or even satisfactory. Third, that operation under local anæsthesia commonly reveals a low-pressure brain with a contused temporal lobe, when a high-pressure bulging brain had been expected. One cannot easily say which of these observations is the most important, but the effects of hypertonic solutions may be singled out. The difference between the results of these injections in head injuries in which the result is often nil as far as return to consciousness is concerned is in the greatest possible contrast to the events which often follow this procedure in brain tumours, that is, in cases where compression cannot be doubted or denied. In the latter the patient commonly becomes an altered being, stupor vanishes, and he arouses sufficiently to allow of valuable observations being made which were impossible

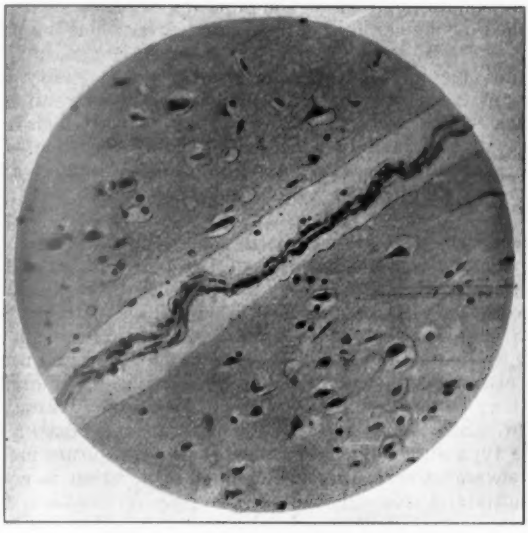

FIG. 7.—Pericellular and perivascular œdema after injury.

before. The effect is due to the removal of that cerebral œdema which accompanies so many tumours (probably the result of histamine), the result being the more dramatic, the more the œdema and the less the tumour. Only occasionally does one achieve a similar result in head injury, not because there is no œdema but because the patient's state is not entirely due to it. In those patients who have a pure œdema with little neural damage a prompt favourable reaction will follow the giving of hypertonic solutions. But in those in whom the cerebrospinal fluid has caused neural disruptions (aided, maybe, by blood vascular leaks) no dramatic improvement will occur or could be expected. In fact if the whole picture of the post-traumatic state were due to a compression relievable by hypertonic solutions, the part played by what we term "physiological decompression" by hypertonic solutions would at this date be so universally accepted that we should not need to inquire into its usefulness. The difference between the results of giving hypertonic solutions to patients with brain tumours

and giving it to those with head injuries is due to the fact that in the latter widespread damage has been induced first and œdema has followed it. If recovery is to be brought about, not only must excess fluid be removed, but the higher neuronal mechanisms which are interrupted must be restored.

The Epiphenomena.—The most important of these are the gross extra- and intra-dural hæmorrhages. I shall only make a few remarks on these in order to open the matter for other speakers. In the first place it should be said that middle meningeal hæmorrhage in its classical form is a rarity, rarer in my experience than the supposedly venous intradural effusions. There is no doubt that it occupies a place in surgical teaching out of all proportion to its frequency in that particular guise. But we do well to expound its stages to students, because they typify certain definite physiological laws. There is, however, to-day little merit in diagnosing a classical extradural hæmorrhage, but there is real merit and satisfaction in discovering one in the severer injuries where consciousness is not regained. In such cases the Hutchinsonian pupil is of the first importance, whilst a contralateral palsy may be picked out by careful bedside study, even when the patient is in some degree of stupor.

That extradural hæmorrhage may be a factor in mortality is made clear by Vance's studies of necropsy material—61 large enough to prove fatal—by compression, in a series of 507 post-mortems. These figures certainly give reason for anxiety if they can be accepted at their face value. However, as we know to our regret, certain of these patients die very quickly before an exploration can be made, whilst others die from associated brain damage after a successful intervention.

Intradural hæmorrhage is commonly described in one form only, the chronic type, giving rise to late symptoms. It ought to be more widely known that it occurs in an acute form, not easily, or at all in some cases, distinguishable in signs from the extradural variety. In Vance's series, again, there were 312 deaths from intradural hæmorrhage, but there is no doubt that these were not local hæmorrhages in the sense that we intend, and that the hæmatoma alone was not truly the cause of death. My own view is that polar contusion is the common cause of such bleeding, that it may be no more than a film of blood or that it may form a massive compressing agent. Sometimes a large hæmorrhage results from a slight contusion, a very favourable type surgically. In other cases there is no more than a film of blood with very severe cerebral injury. Prompt exploration is commonly the best method of diagnosis and although the surgeon may on occasion find that he has operated on a contusion rather than a focal compression, this is a laudable error.

Diagnosis and treatment.—It is of service to have these views in mind when faced with a case of head injury—to recognize that if the patient is definitely stuporous some hours after the injury he certainly has neural damage; "concussion" is the wrong designation for his state. We must learn that the state of stupor and restlessness (Dr. C. P. Symonds has suggested "traumatic stupor" and "traumatic delirium" as terms which suitably describe the states, and these I accept) which we see is due to the fundamental general contusion. General cerebral compression may be present but is not an essential or integral part of the picture, though we must learn to pick out our local compressions so far as possible, and explore them on suspicion.

Treatment by dehydration is at the moment a popular method of treatment of such individuals as we believe to have a general contusion. My own view is that we shouldonly dehydrate in those cases in which we know, by reason of manometric readings, that there is compression. That is to say, the lowering of pressure is not a thing to be indiscriminately employed. The usual methods advocated are: (*a*) Oral and rectal administration of magnesium sulphate; (*b*) intravenous injections of hypertonic solutions, of which the best is 50% glucose in doses of 50 to 100 c.c.,

and (*c*) lumbar puncture. A word of warning is necessary against lumbar puncture as a therapeutic measure. I know of three immediate fatalities after its use in this manner in cases of severe injury see also E. Sachs). It must be cautiously employed, especially on the cyanosed and very ill patient. Lumbar puncture should be reserved for diagnostic refinements—the appearance of the fluid, its red-cell count, and particularly its pressure—no greater quantity being removed than is necessary. We have in hypertonic glucose a much more safe and valuable instrument for treatment when pressure readings indicate it. It will be found of exceptional utility in two classes of cases: First, those in which the patients are recovering consciousness, but are still somewhat unruly, and second, in which the condition does not call for any special anxiety but the patients are drowsy and complain of headache. Pressure readings should, however, be first taken and the glucose, once safely given, may be repeated as symptoms demand. I doubt whether hypertonic solutions have any marked curative effect in the severest head injuries during the first few hours.

ADDENDUM.—A fatality one hour and fifty minutes after the intravenous administration of 50% glucose has recently been reported [Browder].

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Dr. Ritchie Russell: I have had the opportunity, during the past twelve months, of studying the cases of head injury admitted to the surgical wards of the Royal Infirmary, Edinburgh. The condition of the cases on admission, the stages of recovery, the changes in the cerebrospinal fluid and the condition some months after discharge have been the main lines of investigation.

In order to make the records uniform and suitable for statistical study, I have carried out the clinical investigations according to a uniform scheme drawn up in the form of a printed questionnaire.

Records of the acute stage have been made in 200 cases, and I intend, in the short time available, to refer to a few of the observations made.

In attempting to classify the cases according to the degree of damage to the brain, it is convenient to divide those that survive according to the duration of loss of *full* consciousness, keeping the fatal cases in a separate group (Table I). In using this

TABLE I.

Group	Duration of loss of consciousness	Number of cases
A ...	0 to 1 hour	80
B ...	1 to 24 hours	57
C ...	Over 24 hours	47
D ...	Died	16
	Total ...	200

method it is important to realize that the power of speech and intelligible conversation may have returned and yet the patient be quite disorientated and have subsequently no recollection of his actions. Thus the witnesses of an accident often erroneously consider that full consciousness has returned when the victim begins to move or to speak. A fairly accurate estimate of the duration of loss of *full* consciousness may be obtained subsequently from the patient's recollections as to when he became conscious of his surroundings.

Whatever may have been the severity of the injury, the steps towards recovery are somewhat similar in uncomplicated cases, though of course the duration of the stages of recovery varies greatly.

Immediately after the blow the whole of the nervous system may be paralysed, even the respiratory and cardiac movements may cease. If recovery is to occur, the heart and respiration recommence. Involuntary movements of the limbs then occur and the reflexes return. Speech returns with a few words or phrases and movements become more purposeful. Up to this stage it is mainly the lower mechanisms of the brain that are recovering, and the similarity between cases is great. The highest cerebral or mental functions, however, differ in every individual, and the stages of recovery of these functions produce widely different clinical pictures. The mental condition may simulate any of the states seen in alcoholic poisoning. In both conditions, the clinical picture probably depends on the individual psychological structure and balance. Thus the patient may be drowsy or talkative, docile or aggressive, impudent or irritable. He is never reserved, he may tell you his secrets, may be boastful or affectionate, and may even attempt to bribe his attendants to let him out of bed.

Then, comparatively suddenly, he looks around and asks where he is. He has now recovered full consciousness, returns to his normal behaviour and treats those who are looking after him with the customary civility. These changes seem to indicate that the higher functions—of self-consciousness, of control and inhibition—have again taken charge of his behaviour. These are the first to be affected in alcoholic poisoning and are the last to recover after a head injury. They presumably constitute the most sensitive mechanisms in the brain. These stages of recovery may be seen not only in the more severe cases, but also in those in which the individual is unconscious for only a few minutes.

In the series investigated, recollection of the actual blow to the head was never present when consciousness had been lost.

Retrograde amnesia was in most cases of brief duration, as is shown in Table II.

TABLE II.

Approximate Duration of Retrograde Amnesia.

Group	1 or 2 secs.	1 to 30 mins.	Over 30 mins.
A ...	35	4	0
B ...	28	8	1
C ...	6	12	2
Totals ...	69	24	3

This period of memory loss was determined by questioning the patient after full consciousness had returned, as, at an earlier stage, it was found to be of considerably longer duration. While retrograde amnesia of more than a few seconds' duration was seldom found in slight injuries, a longer period was not always present when the injury was severe. Thus in the case of a young man who was unconscious for two weeks, he subsequently remembered colliding with the tar barrel on the road which caused him to be thrown from his motor bicycle.

Physical examination elicited few signs in uncomplicated cases. The pupils were unequal in thirty-eight cases, but the difference in size was usually slight. Abnormality of the plantar reflex is not uncommon in the acute stage and an extensor response was more often found in the severe cases—that is in those cases which were still unconscious when first examined, as is shown in Table III.

TABLE III.

Plantar Reflexes in Cases examined within 24 hours of Accident.

Group	Both flexor	Both extensor	One flexor and one extensor
A ...	46	2	5
B ...	28	3	4
C ...	17	14	3
D ...	3	10	2
Totals ...	94	29	14

A sign which was often present was rigidity of the neck muscles. This was present to a slight degree in thirty-eight cases and to a marked degree in twenty-eight. While it may be due to meningitis, or merely to a bruise in the region of the neck, it is often a sign of subarachnoid hæmorrhage. Thus on thirty-two occasions examination of the cerebrospinal fluid showed it to contain over 1,000 red-blood corpuscles per cubic millimetre, and in all of these patients, with the exception of one who was deeply comatose, the neck muscles showed some rigidity.

Examination of the cerebrospinal fluid was carried out on sixty-seven occasions on forty cases. This was performed with the patient lying on his side with the head and spine at the same level. A fine needle was used, and the pressure of the fluid was in most cases measured with Greenfield's manometer. If care is taken only to penetrate the dura mater on one occasion, and only to pierce it for the least necessary distance, it is my experience that blood contamination from the puncture does not occur except in a degree that can be discerned only on microscopical examination of the fluid. In most cases, both a red and white cell count was carried out within an hour of taking the specimen, and it seems that the red blood-corpuscle count provides an indication of the degree of hæmorrhage which has occurred from the surface of the brain. In all specimens in which blood was present, it was evenly mixed with the spinal fluid, showed no tendency to clot, and after settling the supernatant fluid was yellow in colour.

It is largely through the tearing of small vessels in the pia arachnoid that hæmorrhage into the cerebrospinal fluid occurs. The bleeding is usually most at a point opposite to that at which the injury is received. It seems to me that this so-called contre-coup injury, which is the most constant of all post-mortem findings in these cases, cannot be entirely explained by any wave of force having been applied to the brain at that point. I suggest that the injury is mainly due to the brain

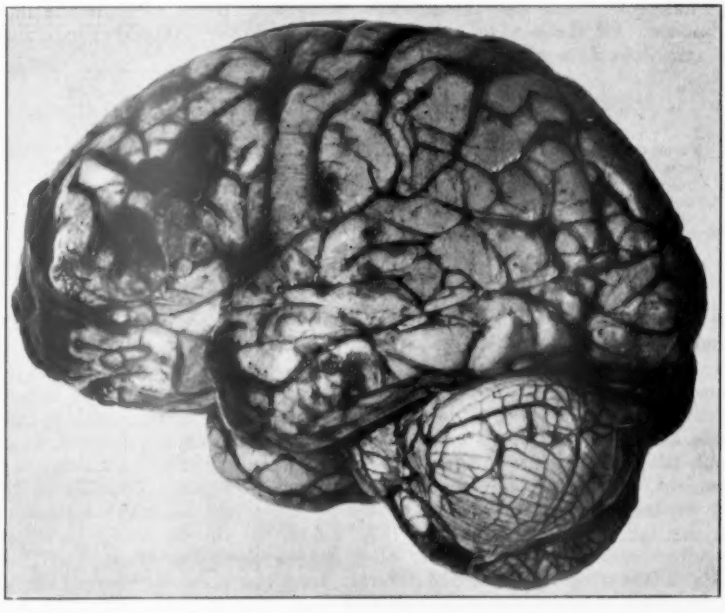

FIG. 1.—Contre-coup injury, showing subarachnoid hæmorrhage.

being torn from its membranes by the force of its own momentum, or of its own inertia according respectively to whether the skull or the object with which it comes in contact has velocity. The place of least resistance to this force is the space between the arachnoid and the pia mater, as this space can be filled by cerebrospinal fluid when the brain is forced away from the skull. The fine bands of fibrous tissue which connect the arachnoid and pia mater are thus torn, with accompanying tearing of pial and cortical vessels. Subarachnoid hæmorrhage is thus a striking feature of a contre-coup injury. The movement of the brain relative to the skull must be greatest at a point opposite to the impact, and hence the damage is most frequently found in this situation.

The following Table shows the relation of the mental state, at the time of the

TABLE IV.

Cerebrospinal Fluid Pressure (Millimetres of Water).

Mental state	0-100	100-200	200-300	Over 300
Normal ...	1	3	10	2
Confused ...	2	3	7	1
Stuporose ...	2	5	4	6
Comatose ...	0	3	0	0
Headache:				
None	1	1	3	1
Moderate ...	0	2	4	0
Severe	1	1	12	4

examination, to the pressure of the cerebrospinal fluid, and also the relation of the latter to the presence or absence of headache. It is apparent from this table that several of the patients who were in a stuporose condition had quite a low pressure, while several with a very high pressure were fully conscious. It is also apparent that in most, but not in all cases of high pressure, there was severe headache at the time of the examination. The Table below shows the relation of the amount of blood-contamination to the mental state. Few cases with much blood in the fluid were conscious and all those with a red cell count of over 100,000 per c.mm. were either stuporose or comatose.

TABLE V.

Blood in Cerebrospinal Fluid (Red Blood-corpuscles per c.mm.).

Mental state	0 to 1,000	1,000 to 100,000	100,000 to 500,000	Over 500,000
Normal ...	11	6	0	0
Confused ...	6	7	0	0
Stuporose ...	2	13	4	0
Comatose ..	0	1	0	2
Headache:				
None	5	2	1	0
Moderate ...	2	1	0	0
Severe	9	8	2	0

The rate at which the blood-cells disappear is shown in figs. 2 and 3. After four or five days there are few red cells to be found, and the fluid becomes clear and yellow-brown in colour. The red cells begin to disappear rapidly from the first day after the accident, so that as in cases H. B. (fig. 2) and J. C. (fig. 3), the rate of their disappearance forms a curve which approximates closely to the straight line. The presence, as in one case, of 50,000 red cells per cubic millimetre, seven days after the accident, probably indicates either that hæmorrhage continued for some days after the accident, or that a delayed hæmorrhage had occurred. The fall in the total protein content of the fluid, as estimated by the micro-Kjaldahl method, parallels closely the fall in the red cell count (figs. 2 and 3). In E. Y. (fig. 2), for example, the protein content by the fifth day after the accident had dropped to 18 mg. per 100 c.c. These observations are of interest from the point of view of the study of the mechanism by which blood is removed from the cerebrospinal fluid, but there is no time to consider them more fully at present.

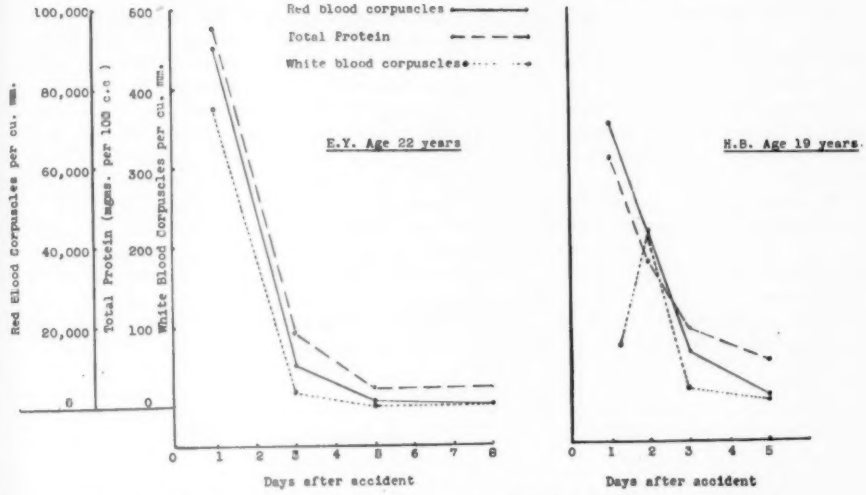

FIG. 2.—To show the rate at which the cerebrospinal fluid usually returns to normal.

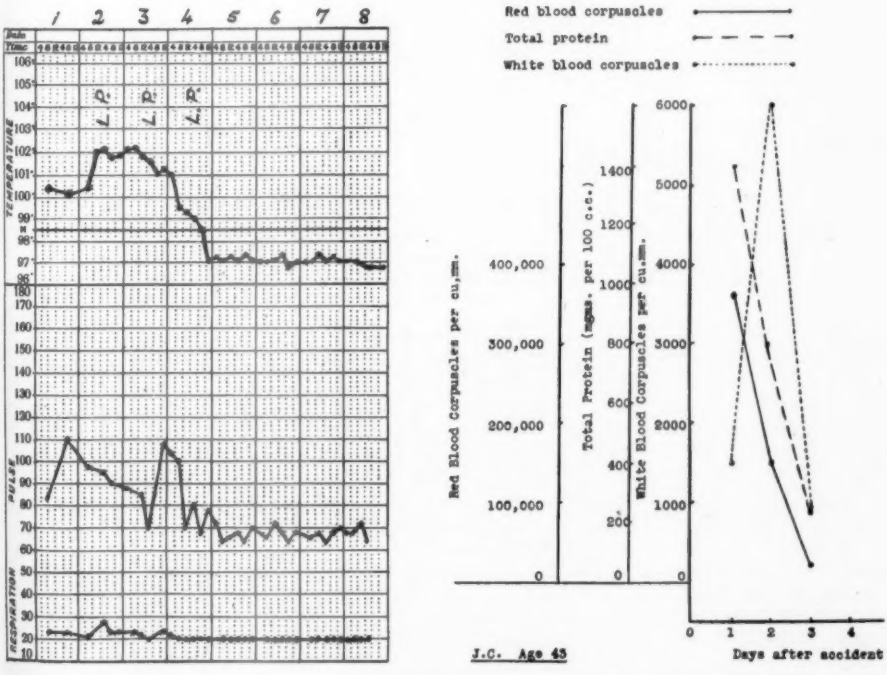

FIG. 3.—Case J. C. Showing the clinical features of meningitis on the second and third days of the illness.

The number of white cells may fall concurrently with the red cells, as in case E. Y., or there may be a temporary rise, as in case H. B. Case J. C., as indicated in fig. 3, showed the clinical features of meningitis on the second and third days of his illness and recovered rapidly following lumbar puncture, so that in the course of twenty-four hours his temperature not only came down to normal, but the white cell-count in the cerebrospinal fluid fell from 6,000 per c.mm. to 800. These changes did not apparently modify the steady disappearance of the red blood-cells and protein.

Hyperglycæmia is present after severe head injury, and correspondingly, the sugar content of the cerebrospinal fluid is raised in the early days after the injury. The drop in the sugar content, which occurs if purulent meningitis is developing, is shown in fig. 4. The rise in the white cell count, which is also shown, was apparent before there was any change in the sugar content.

With regard to treatment, there are many points of uncertainty. No two cases are alike and the value of any therapeutic measure is difficult to assess. Treatment

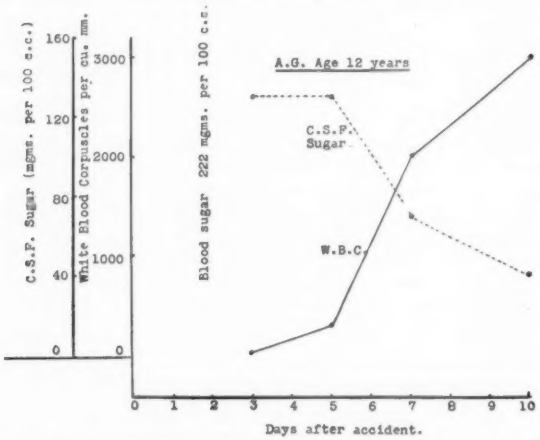

FIG. 4.—Case A. G. Showing the changes in the white corpuscle count and in the sugar content of the cerebrospinal fluid when meningitis was developing.

must therefore be based on the modern knowledge of intracranial physiology combined with one's conception of the mechanism of the changes that occur in the brain as a result of the injury. I have elsewhere[1] drawn attention to difficulties in accepting Trotter's conception of an acute compressive anæmia as the cause of the initial loss of consciousness, and the difficulty of accounting for the condition known as "cerebral irritation" by supposing that it is due to cerebral œdema. It seems to me more probable that the ancient conception of a "commotion" occurring in the nerve elements is the correct explanation of the sudden loss of consciousness. Further, I am inclined to the belief that the condition of so-called cerebral irritation may be more correctly regarded merely as a stage in the recovery of consciousness. The highest cerebral functions having not yet recovered, motor and mental activity runs wild owing to the lack of this control.

At the present time there is a tendency to direct treatment largely, if not entirely, towards reducing intracranial pressure. In the early stages, however, a certain increase

1 "Brain Involvement in Head Injuries." *Transactions of the Medico-Chirurgical Society of Edinburgh*, November 4, 1931.

of pressure is harmless and even desirable for controlling hæmorrhage, and it is only when the increase of pressure is very great or if it becomes persistent, that some attempt may profitably be made to lower it. What seems more important, in the acute stage, is that some means should be taken to prevent violent restlessness, as this is liable, by raising the blood-pressure, to encourage hæmorrhage from torn cerebral capillaries and veins. Morphia is useful for this purpose, and can apparently be given without danger, provided that the patient is allowed to recover from the influence of the drug between each dose, in order that any change in his condition may not be obscured. In the absence of a depressed fracture or of compression of the brain by hæmorrhage, it is doubtful whether there are any indications for operation on the skull in the acute stage.

I wish to express my gratitude to the surgical staff of the Edinburgh Royal Infirmary for allowing me to study the cases under their charge. I have also to thank the Medical Research Council for a grant towards this investigation.

Mr. J. Paterson Ross: I wish to mention three problems which may prove suitable for discussion. (1) *Subdural hæmatoma.*—Chronic subdural hæmatoma has been so well described by Trotter, by Putnam, and before the Section of Neurology this year by Martin, that we are all familiar with the typical clinical picture. The point I wish to stress is the difficulty I have experienced, and I am afraid I am likely to meet it again, of recognizing the atypical case of subdural hæmatoma which is more subacute than chronic. I may illustrate this best by the following case records:—

I.—A printer, aged 40 years, slipped and struck the back of his head on the pavement, on December 26, 1926. He did not lose consciousness, but from the time of the accident he suffered from severe headache, and he vomited five or six times daily. On January 3, 1927, he was admitted to hospital in a state of mild cerebral irritation. The pupils were normal and the optic discs flat. Examination of the central nervous system revealed no abnormal signs. Blood-pressure 118/80. Pulse-rate 68 per minute. A skiagram of the skull showed doubtful linear fracture in the left parietal region, which corresponded roughly to a healed abrasion of the scalp. Lumbar puncture yielded clear cerebrospinal fluid, under considerably increased pressure. January 8, 1927: He was more drowsy and irritable, and the pulse-rate dropped to 60 per minute. There was projectile vomiting. On January 9 he became unconscious, for the first time the pupils were noticed to be unequal, the right being smaller than the left, and within an hour of the appearance of this, the first sign of localizing value, he was dead. This was the fourteenth day after the accident.

Post-mortem examination revealed no fracture of the skull, but there was a large subdural blood-clot overlying the right frontoparietal area.

This was regarded as a case of cerebral contusion, and had subdural hæmatoma been suspected, it is probable that it would have been looked for on the left side (the side of the abrasion and the reported fracture) instead of on the right.

II.—A loader, aged 30 years, admitted unconscious after a street brawl, May 30, 1931. He passed into a state of cerebral irritation, but his pulse rate was usually between 50 and 60, and he remained much the same in spite of saline treatment till June 6, when lumbar puncture was performed and blood-stained fluid, under increased pressure, was withdrawn. The next day he was quite rational and his pulse-rate had risen to 80 per minute. On June 10 he vomited and the pulse-rate fell again to 60, and for the next four days his state of consciousness varied from time to time from normal to extreme drowsiness. These variations became more marked, but on June 15 he became decidedly more drowsy. Up to that time there had been no sign of local lesion of the brain, but on the 15th there was slight weakness of the right side of the face and of the right arm, and the right abdominal reflexes were diminished as compared with the left. For two days there had been a rise of temperature to 99·8° F., and a leucocyte count showed 20,600 cells per c.mm. There was no papillœdema. The diagnosis of cerebral abscess was made and the left cerebral hemisphere was explored widely, with a negative result. The patient died on the nineteenth day after admission, and post-mortem examination showed a large subdural hæmatoma in the right parietal area.

These two cases appear to me to be unlike the classical picture of the man over the age of 50 who, after an insignificant injury, shows no signs of trouble for weeks or months, and then gradually passes into the state of varying degrees of drowsiness, with headache and mental changes, arousing the suspicion of tumour. Of course acute cases have been described, but are usually considered to be rare. It seems to me that the condition must always be borne in mind whenever a case diagnosed as cerebral contusion is progressing unfavourably, and if exploration of one side gives a negative result, the other side must be examined also.

(2) *Local brain lesion without rise in cerebrospinal fluid pressure.*—Seeing that, apart from penetrating wounds, injury to cerebral tissue itself is a much less common cause of paralytic phenomena than pressure of blood-clot upon the affected area, it is natural to suspect intracranial hæmorrhage as the most likely diagnosis in any case of paralysis following a head injury. In the following case the diagnosis of hæmorrhage was considered to be ruled out by the low cerebrospinal fluid pressure, and I bring it forward in the hope of finding out whether others have had similar experience, and whether this is a justifiable deduction to make from observation of the cerebrospinal fluid pressure.

The patient was a boy, aged 19, who was knocked down by a car on May 30, 1927. He was unconscious and, in addition to wounds on the right side of the face and head, he was found to have right-sided hemiplegia with absent abdominal reflexes and an extensor plantar response on the right side. The pupils were normal, the pulse-rate 90, and the systolic blood-pressure 118.

Lumbar puncture showed the cerebrospinal fluid pressure to be 100 mm. of cerebrospinal fluid, and the fluid was slightly and uniformly blood-stained. On this finding it was decided not to operate, and day by day the patient showed slow but steady improvement. On the fourth day he was in a state of cerebral irritation. On the sixth day he spoke indistinctly, and by the twelfth day he was perfectly conscious but with a weak right arm and well marked naming aphasia. In February, 1928, he could walk and talk perfectly, his intellect was unimpaired and he had no headaches, but the right arm was still a little weak. I saw him again at the end of last year when he was perfectly well.

At the first examination the presence of pronounced paralysis suggested intracranial hæmorrhage with pressure on the motor cortex, but the low pressure in the cerebrospinal fluid was interpreted as evidence that the hæmorrhage, if present, could not be extensive, and that symptoms were produced by local injury of the brain tissue by contre-coup.

(3) *Depressed fracture.*—The traditional teaching that all depressed fractures must be operated upon has undoubtedly served a useful purpose, but it is questionable whether it is correct. Nobody will doubt the necessity for operating upon cases of depressed fracture in which there is a penetrating wound of the brain, and it is in such cases, especially if infection supervenes, that epilepsy is not unlikely to be a complication.

I do not, however, believe that operation as a prophylactic against epilepsy is required in all the remaining cases—that is, those without penetration of the brain. These cases may be divided into two groups—those which show evidence of brain injury and those which do not, the injury being presumably the usual cerebral contusion. The objection may be raised that cases of brain injury cannot always be distinguished clinically. But Jefferson was able to show, during the European War, that only five out of fifty-four patients with scalp wounds failed to show neurological symptoms and signs; and it may be fair to assume that if a depressed fracture is present without neurological symptoms and signs there is no appreciable cerebral damage.

Two years ago Mr. Trotter said that for a long time he had felt that the ancient doctrine that, except in the case of the very young, all depressed fractures must be operated upon, was wrong. He had come to hold the opinion that the risk of epilepsy developing from a simple depressed fracture was no greater than the risk of epilepsy developing after an operation including incision of the dura mater, and that it was a

defensible attitude that simple depressed fracture, in the absence of any cerebral symptom, could with safety be left untreated by operation.

The operation performed for simple depressed fracture is elevation of the depressed bone, the dura when undamaged being left intact. Those who practise this operation signify thereby their belief that symptoms are produced by the direct pressure of the bone upon the subjacent cortex. It is possible to imagine that the resolution of an underlying contusion might be delayed by such pressure, but I do not know any evidence to show that this is a factor of importance, and it is improbable that the mere elevation of the bone will have an appreciable effect upon generalized cerebral œdema.

During the past two years I have deliberately refrained from operating upon four patients with depressed fractures. In every case the depression could be readily demonstrated by local examination and radiography, but in none was there any evidence of cerebral injury. The patients are being watched carefully, but so far they have suffered no disability.

Mr. C. H. Fagge (Chairman) said that if he did not misunderstand Mr. Jefferson's meaning, he found himself at variance with the contention that the tendency at present to stress the cerebral rather than the cranial injury was not entirely for the good. To him (the speaker) the changes appeared to have constituted a great advance. In the teaching of students particularly, it was all-important to make them realize that the thing which mattered was the intracranial injury, that the bone might, so to speak, look after itself. That was merely his own view of the question.

He was interested in Mr. Jefferson's remarks about lumbar puncture, because he had been taught that, either as a means of diagnosis, or as a method of relieving intracranial pressure, lumbar puncture was not without danger, and clinical experience had supported that. Could Mr. Jefferson tell him if any clinical signs in a given case could be taken as reliable indications that lumbar puncture was likely to be harmful?

He thought he could supply Mr. Ross with particulars of several cases of depressed fracture which had been treated conservatively, with, so far as he knew, uniformly good results.

Mr. Julian Taylor said he would first like to ask Dr. Ritchie Russell one question. If one could not accept the doctrine that skull deformation with vascular effects was the essential pathological element in concussion, what was meant by "commotion"? Mr. Trotter when writing on the subject decided that the "commotion" had no definite meaning. He would like to know what, in Dr. Russell's opinion, it meant, if the vascular theory were to be discarded. With regard to the relation of cerebral irritation to œdema, he, the speaker, had seen œdema resulting in irritation. One such case was that of a small boy who sustained a blow on the left temporal region. This was explored but the dura was not opened. The boy then showed the ordinary picture of slight irritation, restlessness with drowsiness and headache. On the fourteenth day after the accident he began to have Jacksonian fits on the side of the body opposite to that of the injury. He (the speaker) opened the dura and there saw a small subdural hæmatoma, with the underlying arachnoid raised in blisters, clear evidence of œdema. The decompression effected relieved the fits and the irritation rapidly subsided. Another example was the following: A man, aged 29, fell on the back of his head, suffering practically no concussion; nevertheless in the succeeding days headaches appeared and during the following five weeks he had attacks of drowsiness with increasing headaches. Papillœdema now developed to 5 D. but there were no localizing signs. The diagnosis of a competent neurologist was subtentorial tumour, while that of another was

subdural hæmorrhage, though he was doubtful as to the side. Subdural hæmorrhages were usually alongside the falx and so all one had to do was to bore a hole on either side of it. In this case treacly black blood came out through the needle on one side, nothing on the other. The question of treatment of subdural hæmorrhage was difficult, owing to the fact that the slow growth of such lesions resulted, like that of some cerebral tumours, particularly endotheliomas, in great displacement of the brain, which accommodated itself to the growing tumour. There came a time when the limit of accommodation was reached and then serious symptoms resulted.

In a sense, then, the slower the hæmorrhage, the greater the urgency of operative treatment, and everybody knew the sequence in subdural hæmorrhage of a preliminary attempt at coma with temporary recovery, followed by a second attempt that killed the patient. In the case mentioned, an osteoplastic flap was turned down and a well-developed subdural hæmorrhage with an enclosing membrane containing about half a pint of black fluid was seen. The hemisphere was pushed away towards the middle of the skull and was quite small. The man recovered but during the succeeding days signs of slight, but definite, irritation developed headaches, restlessness and irritability. That this was due to œdema was shown by the lifting of the osteoplastic flap some three-quarters of an inch by the swelling hemisphere as shown in a skiagram. Hæmorrhage was not the cause of this as needles were inserted for exploration around the bone of the flap with negative result. Here then was an example of œdema of the brain resulting in irritative symptoms which had been absent in the earlier stages of the illness when the brain was known to have been compressed.

Mr. Donald Armour said he would like some information from Dr. Riddoch on the underlying pathology of the symptom of photophobia following a head injury. He was not referring to the ordinary photophobia associated with the stage of irritation, but to that which was more or less an isolated symptom. He mentioned two cases in point.

One was that of a prize-fighter who was engaged in a contest in a very brightly-lighted prize ring. He received a blow on the head, but was not marked, or knocked out; he was not even knocked down, but it was evident that something had happened which put him out of business, for he stood with his gloved hands pressed into his eyes, and remained so until he was rushed off to hospital, supposedly the victim of a severe injury. All that the neurologist could find was intense photophobia, and this lasted, gradually diminishing in intensity, for four or five days.

The other case happened in a member of his (the speaker's) own family, who had a closed linear fracture of the frontal bone, a few months ago. When he, Mr. Armour, reached the country hospital where he was, he found him otherwise normal, save that he kept his face buried in the pillows. He had intense photophobia which lasted four or five days, and then gradually disappeared. Otherwise he had been practically symptomless, as far as his head injury was concerned.

Dr. C. P. Symonds said that the first point which had struck him was how convenient it would be to have some terminology which would ensure that all who used the terms were meaning the same thing when speaking of the clinical state in head injury. The tendency of surgeons was to retain "cerebral irritation" as their descriptive term for the state so familiar in severe head injury in the acute stage. The condition had also been referred to as that of "cerebral contusion." He thought that, on the whole, the clinical label was better than the pathological one, especially as so little was still known as to the pathology of the condition. In the Continental and the American literature one found that the terms "traumatic stupor" and "traumatic delirium" were in use, and these appealed to him, rather than "cerebral irritation."

The feature of this discussion which had interested him most was the opinion expressed by those who opened it, that increased intracranial pressure was not the essential cause of what he would prefer to call "traumatic stupor" or "traumatic delirium."

Mr. Jefferson had put clearly three good reasons for supposing that increased intracranial pressure was not the essential cause of the clinical state he had just referred to, and with those reasons he (the speaker) entirely agreed. And to these he would add another, namely, that careful observation of that clinical state would convince anyone who had had a wide experience of the effects of increased intracranial pressure, as exemplified in tumour, hydrocephalus, &c., that traumatic stupor was a different state. In the increased intracranial pressure due to cerebral tumour, &c., the mental state was that of drowsiness with apathy; there was not seen the same motor restlessness, confusion and delirium as in the traumatic cases.

As to the essential basis of traumatic stupor: if it was not pressure, it must be some direct damage to the neural tissues.

He agreed with Mr. Jefferson's observations that the hæmorrhages—gross or petechial—were not so common as one might suppose from the literature. What he regarded as important was that the neuro-pathologists should investigate, in greater detail, the microscopical changes to be seen in those cases. The material for such an inquiry was hard to come by, because the patient who survived for the first two or three weeks after the injury seldom came to autopsy, but there had been published on the Continent one or two careful descriptions of the microscopic appearances seen in such cases, the patient having died of some intercurrent disease after four or five weeks had elapsed. In these, very widespread changes were described, both in the nerve-cells and in the myelin, of a kind which might account better for this peculiar clinical picture than either increased pressure or gross hæmorrhage. If that proved to be the case, then, as far as treatment was concerned, one would return to the conservative methods of prolonged rest and the use of sedatives. And, from his own experience, he could confirm Dr. Ritchie Russell's opinion as to the value of morphia, given in the way that speaker had recommended.

Dr. Russell Brain said that the photomicrographs shown by Mr. Jefferson threw light upon the frequent failure of hypertonic solutions to benefit cases of head injury. Weed and his fellow-workers had shown that their administration to normal animals caused a fall in intracranial pressure not only by diminishing the formation of the cerebrospinal fluid but also by promoting its absorption into the blood-stream through the perivascular spaces. Mr. Jefferson had shown that these absorptive channels were damaged in cases of head injury, and he (Dr. Brain) doubted whether it was wise to throw a strain upon them by administering hypertonic solutions.

Dr. Riddoch, in reply, said that the main lesson to be learned from the discussion was that knowledge of the pathology, symptomatology and treatment of head injuries was still imperfect. It was to be hoped that interest in this important subject would be revived.

To answer Mr. Armour's question concerning photophobia was difficult. Along with restlessness, irritability and headache, photophobia often remained for a considerable time after consciousness had been regained, and was sometimes associated with little more than pain behind the eyes.

Dr. Ritchie Russell, in reply to Mr. Julian Taylor, said that he had no precise idea of what the term "commotion" implied. It was, however, probable that loss of consciousness was due to the effect of jarring sustained by the nerve elements. He could only presume that the disturbance was molecular in nature.

Cases had been described of cerebral œdema associated with the clinical picture of so-called cerebral irritation. While there was no doubt that circulatory disturbances in the brain could cause great mental disturbance, the clinical picture "cerebral irritation" was absent in other conditions associated with cerebral œdema, such as cerebral tumour and uræmia. He could not see that there was any evidence that the irritable state of these patients was to be attributed to an irritative condition affecting the nerve elements.

Mr. Jefferson, in reply to the President, explained that what he had meant was that at the present time the profession was in a better position to tackle the problem of fracture when the other (intracranial damage) had been finished with; that in the light of modern knowledge he was not satisfied with all the explanations. That was all he had wanted to throw out in the way of criticism.

With regard to lumbar puncture and its danger, the point he had to make in that connection was, that the withdrawal of fluid was dangerous unless one had first taken a reading. To draw off a quantity of highly blood-stained fluid from a deeply stuporose—and, particularly, a deeply cyanosed—patient, was, he considered, likely to court disaster. It was the presence of deep cyanosis and stupor which would make him feel very hesitant about drawing off fluid without knowing what the pressure was. In these cases the deaths must be due to the same thing as in tumour, i.e., foraminal herniation.

With what Dr. Symonds said he was in entire agreement; the profession would do well to adopt the terminology suggested.

The other chief point which emerged was that raised by Mr. Paterson Ross about pressure readings. This year there occurred the case of a man who lay unconscious several days, with a very slow pulse-rate, aphasia, some stupor mixed with the aphasia, and weakness of his right arm. He, the speaker, measured the cerebro-spinal fluid pressure, which was found to be 10 c.c. of water. His remark to the house-surgeon was, "We have solved one problem, we know he has no clot, and we can watch him with confidence." But in two days the patient had become worse, and was more deeply unconscious. He, Mr. Jefferson, explored, and found extradural hæmorrhage over the lower part of the motor area on the left side. In spite of the removal of the clot the patient did not make an immediate recovery. It was clear afterwards that there were two conditions: he had a small clot, which was 1 in. thick, though not greater in diameter than 1½ in. But he did not show immediate improvement because there was an underlying contusion of the brain. After four weeks of semi-stupor, recovery was complete.

The question of acute subdural hæmatoma was a very difficult one. He agreed with Mr. Julian Taylor that the surgeon must explore both sides; that was the way to find out. He had had a case in a girl, aged 8 years, who when admitted to hospital was thought to have subdural hæmorrhage. He opened the wrong side; she was too ill to allow of the other side being opened. Post mortem there was found to be an enormous hæmorrhage on the opposite side. The time which elapsed between the accident and the death (including the operation) was only thirty-six hours. He might have saved the patient if her state had allowed him to carry out what had been his usual practice since, namely, opening both sides.